CLINICAL PROFILE OF IDIOPATHIC INTRACRANIAL HYPERTENSION IN A TERTIARY EYE CARE CENTRE IN SOUTH INDIA

DISSERTATION SUBMITED FOR

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CERTIFICATE

This to certify that this dissertation entitled **"CLINICAL PROFILE OF IDIOPATHIC INTRACRANIAL HYPERTENSION IN A TERTIARY EYE CARE CENTRE IN SOUTH INDIA"** is a bonafide work done by **Dr.G.CHITRA** under our guidance and supervision in the Neuroophthalmology department of Aravind Eye Hospital and Post Graduate Institute of Ophthalmology, Madurai during the period of her post graduate training in Ophthalmology for May 2013-April 2016.

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DECLARATION

I, Dr.G.Chitra hereby declare that this dissertation entitled, **CLINICAL PROFILE OF IDIOPATHIC INTRACRANIAL HYPERTENSION IN A TERTIARY EYE CARE CENTRE IN SOUTH INDIA",** is being submitted in partial fulfilment for the award of M.S. in Ophthalmology Degree by the Tamilnadu Dr.MGR Medical university in the examination to be held in April 2016.

I declare that this dissertation is my original work and has not formed the basis for the award of any other degree or diploma awarded to me previously.

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INTRODUCTION

Idiopathic intracranial hypertension is a neurological disorder characterized by elevated cerebrospinal fluid pressure without a known cause. It is more common in obese women of child-bearing age[1] and is uncommon in the non-obese, men, elderly and very young children.Though the annual incidence of IIH in general population is low (0.9/100,000), the incidence in women between 20 and 44 years of age is 3.5/1,00,000 and 19/1,00,000 in women who are 20% over weight.[2]

The most common presenting complaints are headache(76%),reduced visual acuity(48%),transient obscuration of vision(48%),nausea and vomiting (30%), and diplopia (8%)[1].The most common visual field defect is an enlarged blind spot. Papilledema is the ophthalmologic hallmark of Idiopathic intracranial hypertension Neuroimaging is usually normal. Diagnosis is based on Modified Dandy criteria. The neurological examination will be normal apart from papilledema and a 6^{th} nerve palsy which is a false localizing sign.

The commonly associated risk factors are Hypothyroidism, Hyperthyroidism, Obesity, Pregnancy, Anaemia, Hypertension, Obstructive sleep apnoea, exposure to exogenous drugs like Tetracyclines, Corticosteroids,

Oral contraceptives, Vitamin A intoxication, nalidixic acid and topical isotretinoin. Increased intracranial venous pressure related to stenosis of the cerebral venous sinuses is another proposed causal mechanism of idiopathic intracranial hypertension[3] .The treatment includes Weight reduction, Repeated lumbar punctures, Carbonic anhydrase inhibitors and Diuretics. Surgical treatment includes Optic nerve sheath fenestration, Bariatric surgery, Lumbo-peritoneal Shunting procedures and Venous sinus stenting. The most important metrics when following IIH patients are formal perimetry and fundus examination, often augmented by stereo optic disc photographs.

ANATOMY OF OPTIC NERVE

It is the second cranial nerve. Each optic nerve starts from the optic disc and extends upto the optic chiasma, where the two nerves meet. It is the backward extension of the nerve fibre layer of the retina which consists of the axons originating from the ganglion cells[4]

PARTS OF THE OPTIC NERVE

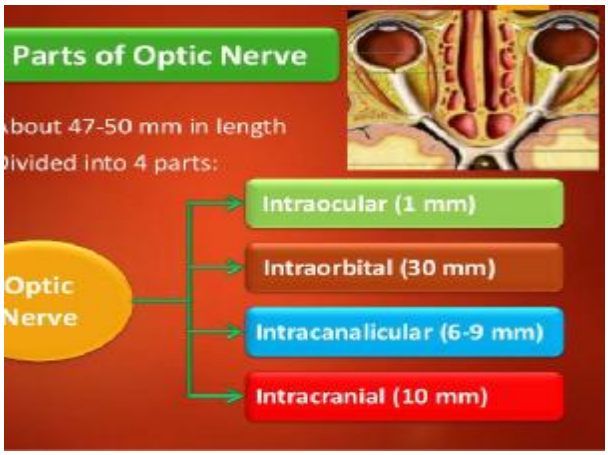
The optic nerve is about 47-50mm in length and can be divided into 4 parts:

a. Intraocular part (1mm)

b. Intraorbital part (30mm)

c. Intracanalicular part (6-9mm) and

d. Intracranial part (10mm)



ANATOMY OF OPTIC NERVE HEAD (INTRAOCULAR PART) (OPTIC PAPILLA, OPTIC DISC)

The optic nerve head is the distal part of the optic nerve[4]. It extends from the retinal surface to the myelinated part of the optic nerve just behind the sclera and lamina cribrosa. The optic nerve head is so called because of its three-dimensional structure.

COMPOSITION

Nerve fibres arise from the ganglion cell layer and converge upon the ONH. Connective tissue support is provided by the glial cells and astrocytes. The axons at the superior and inferior poles have less structural support. The axons exit the globe through a fenestrated scleral canal, the lamina cribrosa. Optic nerve diameter expands to about 3mm once the axons become myelinated in the retrolaminar part.

The optic cup is pale in color due to the visibility of the lamina cribrosa and presence of connective tissue. The lamina cribrosa has fenestrations which appear as dots. The normal optic disc is vertically oval while the cup is horizontally oval and slightly displaced superiorly. The inferior rim is the thickest. followed by nasal, temporal being thinnest.

The optic nerve head exhibits three zones[4] namely,

1. Surface nerve fibre layer
2. Prelaminar region
3. Lamina cribrosa
4. Retrolaminar region

SURFACE NERVE FIBRE LAYER

It contains predominantly of axonal nerve fibres of the ganglion cells along with interaxonal glial tissue.

PRELAMINAR REGION

The structures at this level are neurons and a significantly increased quantity of astroglial tissue.

LAMINA CRIBROSA

It is a fibrillar sieve like structure made up of fenestrated sheets of scleral connective tissue lined by glial tissue. It bridges the posterior scleral foramina or the scleral canal. The bundles of optic nerve fibres leave the eye through these fenestrations.

RETROLAMINAR REGION

This area is characterised by a decrease in astrocytes and the acquisition of myelin that is supplied by oligodendrocytes. The addition of myelin sheath nearly doubles the diameter of the optic nerve (from 1.5 to 3.0 mm) as it passes through the sclera.

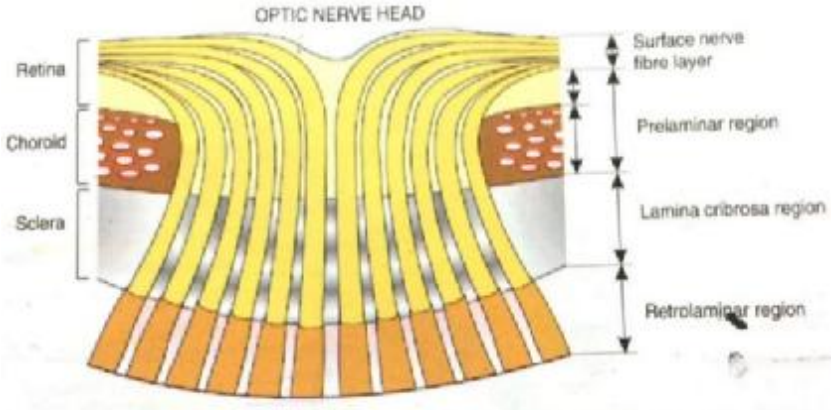


Fig. 6.13. Schematic drawing of optic nerve head.

INTRAORBITAL PART

Intraorbital part of the optic nerve is from behind the globe to the optic foramen. This part is sinuous. Important relations of this part are:

1. The optic nerve in the orbit is covered by duramater, arachnoid and piamater.

2. The central retinal artery along with the accompanying vein crosses the subarachnoid space to enter the nerve on its inferomedial aspect about 10mm from the eyeball.
3. Near the optic foramen, the optic nerve is closely surrounded by the annulus of Zinn
4. The long and short ciliary nerves and arteries surround the optic nerve before these enter the eyeball.

INTRACANALICULAR PART

1. This part is closely related to the ophthalmic artery which crosses the nerve inferiorly from medial to lateral side in the dural sheath and then leaves the sheath at the orbital end of the canal.

INTRACRANIAL PART

1. This part of the optic nerve, about 1 cm in length It is ensheathed in piamater, but receives arachnoid and dural sheaths at the point of its entry into the optic canal[4].

MENINGEAL SHEATHS OF OPTIC NERVE

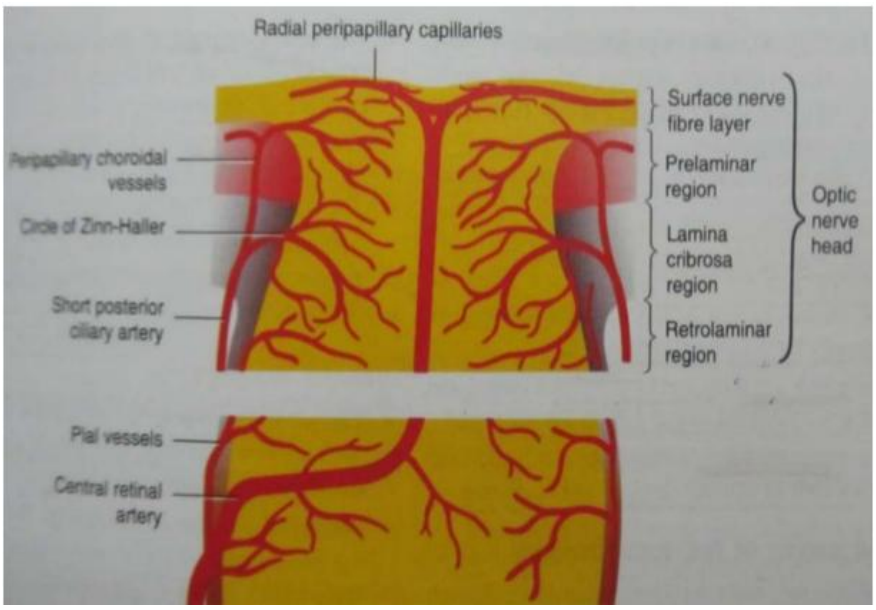
1. The intracranial part of the optic nerve is covered by piamater only, while the intracanalicular and intraorbital parts of the nerve have three coverings: the piamater, arachnoid and duramater.

2. The meningeal sheaths and the subarachnoid and the subdural spaces around the optic nerve are continuous with those of the brain[4].

BLOOD SUPPLY OF OPTIC NERVE HEAD

INTRAOCULAR PART

a. The surface nerve fibre layer is mainly supplied by the capillaries derived from the retinal arterioles, which anastomose with vessels of the prelaminar region.
b. The prelaminar region is supplied by peripapillary choroidal plexus.
c. The lamina cribrosa region is also supplied by the ciliary vessels which are derived from the short posterior ciliary arteries and arterial circle of Zinn-Haller.
d. The retrolaminar region is supplied by both the ciliary and retinal circulation with the former coming from recurrent pial vessels. The central retinal artery provides centripetal branches from the pial plexus and also centrifugal branches.



BLOOD SUPPLY OF OPTIC NERVE HEAD

INTRAORBITAL PART

The intraorbital part of optic nerve is supplied by two systems of vessels-a periaxial and an axial:

a. The periaxial system of vessels supplying this part of optic nerve is derived from the six branches of internal carotid artery namely: ophthalmic artery, long posterior ciliary arteries, short posterior ciliary arteries, lacrimal artery and central artery of retina before it enters the optic nerve and circle of Zinn.
b. The axial system of vessels supplying the axial part of the optic nerve is derived from:

1) the intraneural branches of the central retinal artery
2) central collateral arteries which come off from the central retinal artery before it pierces the nerve and
3) central artery of optic nerve.

INTRACANALICULAR PART

The nerve within the optic canal is supplied only by the periaxial system of vessels. The pial plexus in this part is fed mainly by branches from the ophthalmic artery.

INTRACRANIAL PART

Intracranial portion is exclusively supplied from the periaxial system of vessels. The pial plexus here is contributed by 4 sources:

1) branches from the internal carotid artery either directly or through the recurrent branch of anterior superior hypophyseal artery(supply the inferior aspect of the optic nerve containing lower retinal fibres)
2) branches from anterior cerebral artery (supply the superior aspect of the optic nerve containing upper retinal fibres),
3) small recurrent branches from the ophthalmic artery and,
4) the twigs from the anterior communicating artery.

VENOUS DRAINAGE

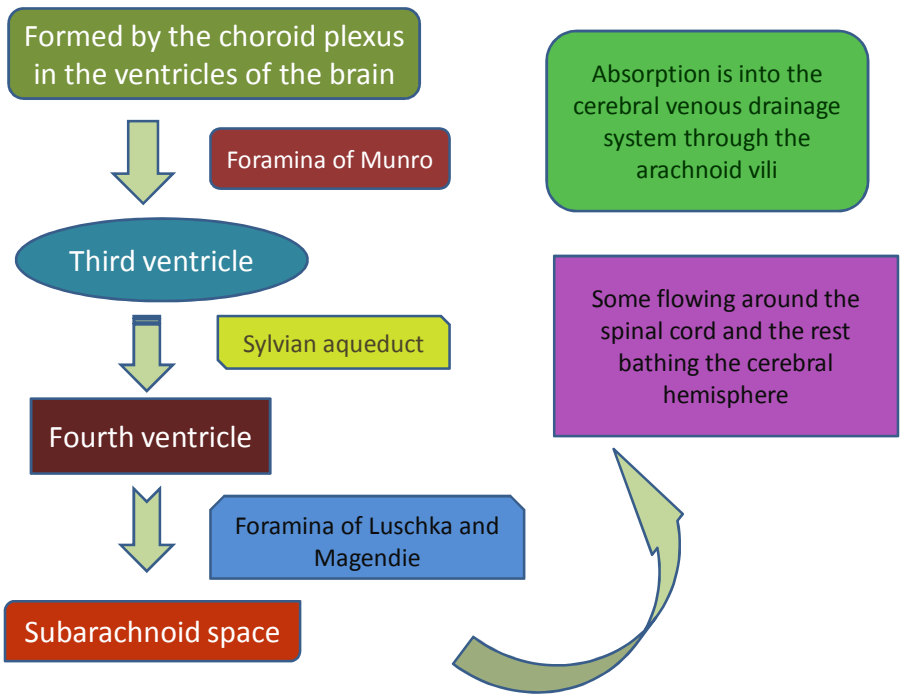
A. The venous return in the optic nerve head is primarily by the central retinal vein.

B. The orbital part is drained by peripheral pial plexus and also by central retinal vein in the distal part.

C. The intracranial part is drained by the pial plexus which ends in anterior cerebral and basal vein.

- Autoregulation
- Presence of blood-brain barrier[4].

CIRCULATION OF CEREBROSPINAL FLUID



CEREBRAL VENOUS SINUSES

Dural venous sinuses are venous channels located intracranially between the two layers of duramater (endosteal layer and meningeal layer). Unlike other veins in the body they run alone, not parallel to arteries. They are valveless, allowing for bidirectional blood flow in intracranial veins. They form the major drainage pathways from the brain.

The main dural venous sinuses are:

UNPAIRED

1. Occipital sinus
2. Straight sinus
3. superior sagittal sinus
4. inferior sagittal sinus
5. basilar venous plexus
6. anterior intercavernous sinus
7. posterior intercavernous sinus

PAIRED

1. sigmoid sinus
2. transverse sinus

3. cavernous sinus
4. inferior petrosal sinus
5. superior petrosal sinus
6. sphenoparietal sinus
7. petrosquamous
8. middle meningeal

TRANSVERSE SINUS

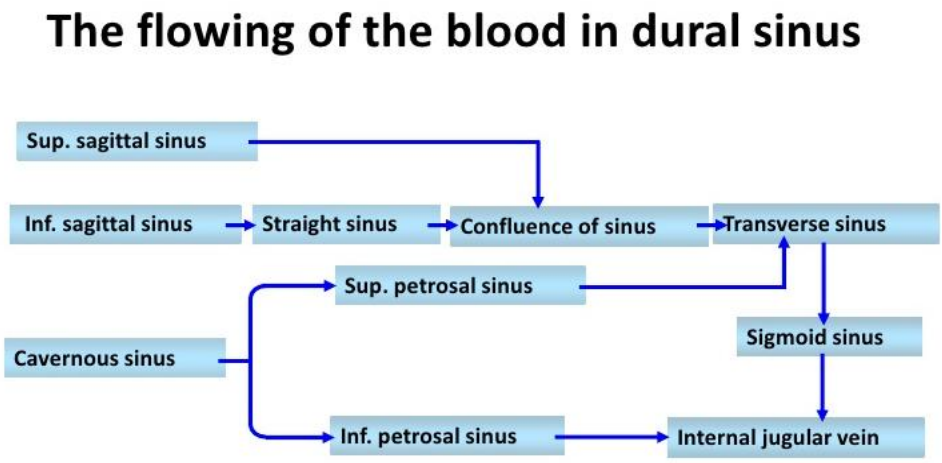
The superior sagittal sinus, the occipital sinus and the straight sinus are drained by transverse sinus and empties into the sigmoid sinus which in turn ends in the jugular bulb

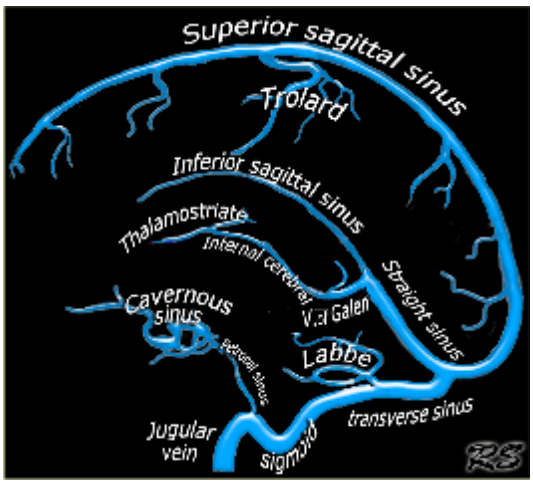
Variant anatomy

- 39% Hypoplasia of left sinus
- 20% Aplasia of left sinus
- 31% Symmetrical
- 6% Aplasia of right sinus
- 4% Hypoplasia of right sinus

SIGMOID SINUS

The right and left sigmoid sinuses are continuation of the corresponding transverse sinuses and become the sigmoid sinus. Here the sinus receives the superior petrosal sinus. It ends in the jugular bulb in the posterior half of jugular foramen.





AXOPLASMIC TRANSPORT

This Process is responsible for the supply of the nutrients within the axons.

- Axoplasmic Flow
    - Antegrade flow
    - Retrograde flow
    - Fast & slow components

Constant antegrade and retrograde flow of materials is necessary for the maintenance of the axons.

NORMAL AXOPLASMIC FLOW

Slow component

- 1 – 3 mm per day
- Driven by the peristaltic wave

Rapid component

- Pass through disc quickly and evenly
- 400 mm per day

FACTORS INFLUENCING AXOPLASMIC FLOW

- Intra axonal pressure
- Intra ocular pressure
- Optic Nerve Tissue pressure (ONTP)
- CSF pressure

CAUSES OF RAISED INTRAOCULAR PRESSURE

Blood

- Hemorrhage
- Obstruction of venous outflow (cerebral venous thrombosis)

Brain

- Mass lesions (tumor, abscess)
- Cerebral edema (trauma, Hypoxic ischemic encephalopathy, electrolyte abnormalities, metabolic abnormalities, meningitis)

CSF

- Increased production (choroidal plexus papilloma-rare)

- Decreased drainage (communicating/non-communicating hydrocephalus, shunt malfunction)

Idiopathic

- Idiopathic intracranial hypertension (Pseudo-tumor Cerebri)

PAPILLOEDEMA

Papilledema is a passive, non-inflammatory, hydrostatic edema of the optic nerve head due to increased intracranial pressure.

It is almost always bilateral and asymmetrical[5].

NORMAL OPENING PRESSURE ON LUMBAR PUNCTURE

INFANTS - <80 mmH2O

CHILDREN - <90 mmH2O

ADULTS - <210 mmH2O[5]

DIAGNOSIS OF RAISED INTRACRANIAL PRESSURE

1. **Headaches-**more severe in the morning, worsening progressively. Intensifies with head movement.
2. **Sudden nausea and vomiting**, often projectile, may partially relieve the headache.
3. **Deterioration of consciousness** may be slight, with drowsiness and somnolence. Dramatic deterioration of consciousness is indicative of brainstem distortion with tentorial or tonsillar herniation and requires prompt attention.

4. **Visual symptoms**

   a. **Transient Obscurations** lasts usually for five seconds, rarely exceeds 30 seconds at irregular intervals

   b. **Horizontal diplopia** due to abducent nerve paresis caused by stretching of one or both 6th nerves over the tip of petrous temporal bone; this is therefore a false localizing sign[5].

   c. **Visual loss** occurs late with secondary optic atrophy due to long-standing papilledema.

5.Investigations

MRI, CT and B-SCAN show an enlarged optic nerve diameter.

VISUAL FIELD DEFECTS COMMON IN PAPILLEDEMA

1. Enlargement of blind spot
2. Concentric contractions
3. Relative scotoma first to green and red
4. Complete blindness
5. Homonymous hemianopia
6. Central and arcuate scotomas
7. Infero-nasal quadrant -most commonly involved

THEORIES OF PAPILLEDEMA

EARLIER THEORIES

1. Non-mechanical
2. Inflammatory by Gowers and Leber
3. Vasomotor theory by Kornder

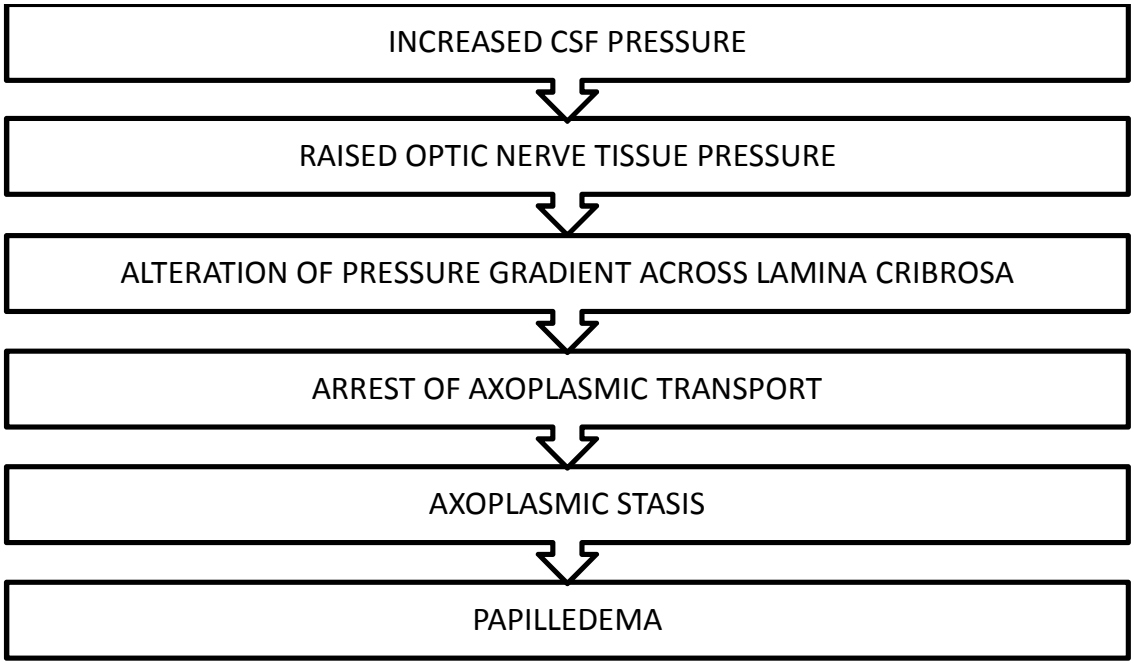
MECHANICAL THEORY

1. Venous obstruction
2. Obstruction of tissue fluid
3. Forcing of CSF into optic nerve

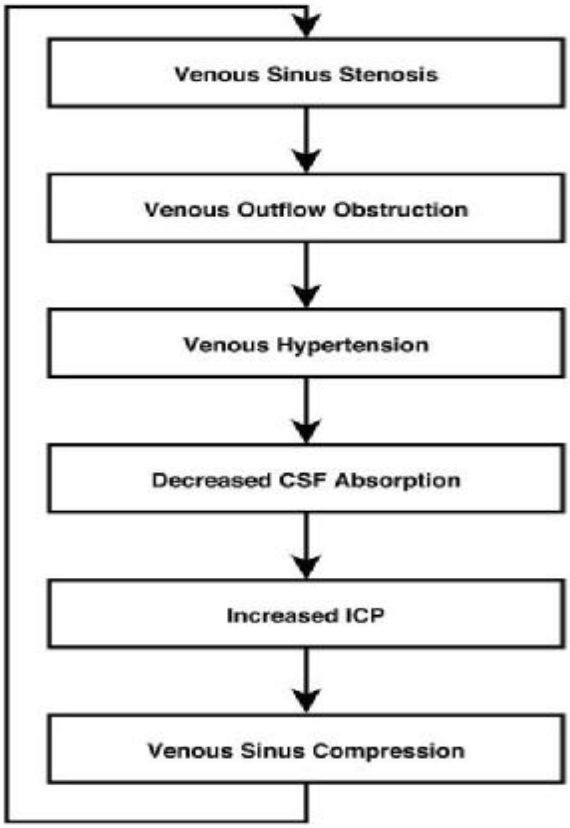
MODERN THEORY

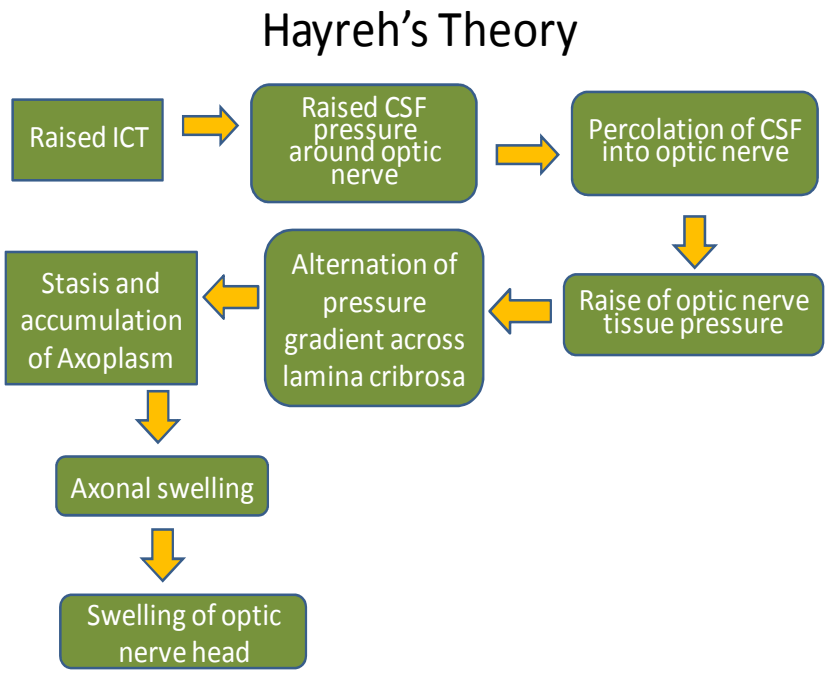
1. Hayreh's Theory of axoplasmic stasis (most accepted theory)

MECHANISM OF PAPILLEDEMA



MECHANISM BY WHICH VENOUS STENOSIS CAUSES PAPILLEDEMA





FUNDUS SIGNS IN OPTIC DISC EDEMA

MECHANICAL SIGNS:

1. **Optic nerve head swelling**

   1mm disc elevation causes+3D refractive change.

2. **Blurring of optic disc**

3. **Filling of the physiological cup:**

   It occurs due to axons crowdind leading to narrowing of the optic cup.

4. **Edema of the peripapillary nerve fibre layer**[6]

5. **Retinal or choroidal folds:**

These occur in the peripapillary area and the posterior pole. Linear lines which develop are also referred to as Paton's lines[6].

VASCULAR SIGNS

Disc hyperemia

- It is due to an increased vascularity of the disc and opening up of the smaller blood vessels on the surface of the disc.
- This leads to an increase in the Kestenbaum's number (number of small vessels traversing the optic disc)[6]. This becomes more than 12 in disc hyperaemia.

Vascular congestion

(Venous dilatation and tortuosity):

It is due to reduction in the venous outflow and increased pressure within the venules.

Peripapillary haemorrhages:

These occur due to rupture of small blood vessels.

Exudates in the disc or peripapillary area:

This occurs due to leakage of proteins and lipids from congested blood vessels.

Nerve fibre layer infarcts:

- This appears as areas of pale edema in the peripapillary areas.

GRADING OF PAPILLEDEMA

EARLY PAPILLEDEMA

1. Disc swelling
2. Disc hyperemia
3. Blurring of optic disc margins.
4. Blurring of the peripapillary nerve fibre layer
5. Dilatation of retinal veins
6. Absence of spontaneous venous pulsations

ESTABLISHED PAPILLEDEMA

1. Increase in disc swelling(2 to 6 dioptres)
2. Peripapillary chorio-retinal folds
3. Venous engorgement, splinter hemorrhages and microaneurysms

4. In advanced cases, amyloid bodies may develop over the disc surface.

CHRONIC PAPILLEDEMA (Vintage papilledema)

1. Resolved hemorrhages, exudates and edema over the disc.
2. The disc hyperaemia and haemorrhages reduce.
3. Disc edema persists in this stage for months to years without any change.
4. Classically, it appears as “CHAMPAIGNE CORK”.
5. The disc eventually becomes dirty gray and pale in color.
6. Optociliary shunts and drusen like deposits may be present on the disc.

ATROPHIC DISC EDEMA

1. Grey white pallor of disc with blurred margins
2. Attenuated sheathed vessels
3. The resolving edema may leave “WATER MARKS”.
4. Gliosis on the disc surface and margins

HISTOPATHOLOGICAL FINDINGS IN PAPILLEDEMA[6]

1. Edema and vascular congestion.
2. Peripapillary haemorrhages.
3. Obliteration of the optic cup.
4. Disc elevation into the vitreous cavity.

5. Engorgement and tortuosity of the small blood vessels.
6. Accumulation of the extracellular fluid in and anterior to the retinal lamina cribrosa
7. Enlargement of the subarachnoid space, with stretching.
8. Engorgement of axons in the prelaminar portion.

   Electron microscopy of these axons shows:

   - Axonal swelling and accumulation of mitochondria.
   - Mitochondrial swelling and disruption.
   - Disruption of fascicles of the microtubules.
9. Peripapillary sensory retinal changes include:
   - Displacement of the retina away the disc.
   - Buckling of the outer layers of the retina.
   - Displacement of the rods and cones away from their anchor near Bruch's membrane.
   - Serous retinal detachment in the peripapillary area may occur.
10. Chronic disc edema is characterised by degenerative and fibrotic changes.
11. Established papilledema shows secondary optic atrophy with subsequent gliosis and fibrotic.

FRISEN'S GRADING SYSTEM[2]

a. Stage 0:

1. Mild nasal elevation of nerve fibre layer
2. A portion of major vessels obscured in upper pole.

b. Stage 1: Very early papilledema

- Obscuration of nasal border of disc
- No elevation
- Disruption of nerve fibre layer
- Concentric or radial retinal halo

c**. Stage 2: Early papilledema**

- obscuration of all borders
- elevation of nasal border
- complete peripapillary halo

d. **Stage 3: Moderate papilledema**

- Obscuration of all the borders
- Increased diameter of optic nerve head
- Obscuration of one or more segments of major blood vessels leaving the disc

e. **Stage 4: Marked papilledema**

- Elevation of the entire nerve head
- Obscuration of all the borders
- Peripapillary halo
- Total obscuration on the disc of a segment of major blood vessels

f. **Stage 5: Severe papilledema**

- Dome shaped protrusions representing anterior expansion of optic nerve head
- Peripapillary halo
- Total obscuration of a segment of major blood vessels
- Obliteration of optic cup
- Obscuration of all the borders

PSEUDOPAPILLEDEMA

Disc appearance resembling disc edema, in the absence of a true disc edema[6].

Features of pseudopapilledema:[6]

a. Absence of central cup but presence of venous pulsation.
b. Vessels arise from the central apex of the disc.

c. Anomalous branching of the vessels on the disc and increased number of major disc vessels.
d. Disc transillumination shows drusens.
e. Disc margins show irregular outline Absence of superficial capillary telangiectasia, haemorrhages, exudates and cotton wool spots.
f. Absence of peripapillary retinal folds.
g. Lack of fluorescein leakage from disc vessels.
h. Drusen shows autofluorescence in FFA prior to dye injection and stains in the late stages of the angiogram.

DD FOR PSEUDOPAPILLEDEMA

1. Small hypermetropic disc
2. Optic nerve head drusens
3. Medullated nerve fibres

IDIOPATHIC INTRACRANIAL HYPERTENSION

INTRODUCTION

Idiopathic intracranial hypertension is a syndrome characterised by elevated intracranial pressure without ventriculomegaly or mass lesion, and with normal cerebrospinal fluid composition that usually occurs in obese women of child-bearing age. It is a disorder of elevated CSF pressure. It is often referred to as "Pseudotumour cerebri"[7]

Quinke in 1897 reported the first cases of IIH. It was named Pseudotumour Cerebri in 1904. Initially the term Benign intracranial hypertension was coined by Foley in 1955.

The underlying cause is reduced absorption of cerebrospinal fluid. The ventricular system is normal without any deformity and obstruction. The neuroimaging is normal except for raised CSF pressure.

The CSF pressure is above 200mm of water in the non-obese and above 250mm of water in the obese.

The peak incidence is in the 3rd decade of life, especially obese females in the reproductive age group.

The most common presenting symptoms of raised intracranial pressure are headache, pulsatile tinnitus, transient obscuration of vision, pain behind the eyes, double vision and loss of vision.

Signs are diplopia due to abducent nerve paresis and papilledema resulting in loss of sensory visual function.

Most Common visual field defect is an enlarged blind spot.

This satisfies the modified Dandy criteria for IIH.

Neuroimaging signs include empty sella syndrome, lateral or transverse sinus stenosis, flattening of globe and unfolding of optic nerve sheaths.

CRITERIA FOR DIAGNOSIS (MODIFIED DANDY CRITERIA)[8]

1. "Signs and symptoms of increased intracranial pressure headaches, nausea, vomiting, transient obscurations of vision, papilledema).
2. No localising, focal neurologic signs, except unilateral or bilateral sixth nerve paresis.
3. Cerebrospinal fluid opening pressure ≥ 25 cm, but without cytologic or chemical abnormalities.
4. Normal neuroimaging results adequate to exclude cerebral venous thrombosis, i.e., magnetic resonance imaging of the brain, often with

additional sequences (computed tomography or magnetic resonance venography).”

EPIDEMIOLOGY

The annual incidence in general population is 0.9/1,00,000 and 3.5/1,00,000 females. In obese women aged 25 to 40 years, the incidence is 19/1,00,000. The female to male ratio is 2:1. More than ninety percent of IIH patients are obese and more than ninety percent are women of child-bearing age[2].

ETIOLOGY

1. Usually idiopathic in over 90% cases
2. In 10% cases, in men and non-obese,

   Obstruction to cerebral venous drainage

   Endocrine and metabolic dysfunction

   Exposure to exogenous drugs

   Systemic illness

ASSOCIATIONS OF IDIOPATHIC INTRACRANIAL HYPERTENSION[6]

1. **Obstruction or impairment of cerebral venous drainage:**

   Tumours

   Septic thrombi

   Radical neck dissection

2. **Endocrine and Metabolic Dysfunction:**

   Elevated oestrogen levels

   During pregnancy (especially the 2^{nd} and the 5^{th} month) there is decrease in the adrenal corticoids and increased oestrogen.

Hypoparathyroidism and associated hypocalcemia interferes with transport of CSF through arachnoid granulations.

3. **Exogenously administered agents:**

Systemic steroid therapy leads to suppression of the adrenal cortex.

Antibiotics (tetracycline, nalidixic acid)

Anti-inflammatory agents: (Indomethacin, Ketoprofen)

Vitamin A - hypervitaminosis

Lead encephalopathy causes cerebral edema and raised ICT.

4. **Systemic illness:**

Meningitis and encephalitis lead to blockage of ventricular system.

Status epilepticus leads to cerebral hypoxia and cerebral edema.

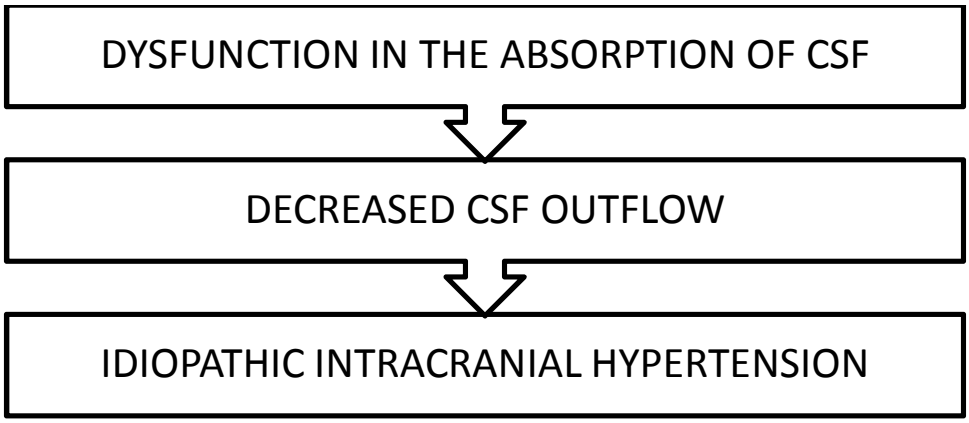
Vascular Hypertension.

Thrombocytopenic purpura.

Chronic respiratory insufficiency, hypercapnia, reduced blood oxygen, polycythemia, elevated venous pressure and ICT.

5. **Familial Benign intracranial hypertension**
6. **Obstructive sleep apnoea**
7. **Iron deficiency anemia**
8. **Thyroid replacement therapy**

PATHOGENESIS



CLINICAL FEATURES

SYMPTOMS

1. Headache
2. Transient visual obscuration
3. Pulsatile tinnitus
4. Flashes of light
5. Double vision
6. Loss of vision
7. Nausea and vomiting

HEADACHE:

1. Usual presenting symptom

2. Severe daily pulsatile
3. May awaken the patient from sleep

TRANSIENT VISUAL OBSCURATIONS

1. Usually 5 seconds rarely exceeds 30 seconds
2. Followed by recovery of vision to baseline
3. Monocular or binocular
4. They are due to transient compression or ischemia of the optic nerve head

PULSE-SYNCHRONOUS TINNITUS

1. Pulsatile intracranial noises often unilateral
2. The noise is abolished by jugular compression or head turning ipsilateral to the sound .

VISUAL LOSS

1. Relatively mild at presentation
2. Best corrected visual acuity ranges from 6/6 to perception of light.

OCULAR MOTILITY DISTURBANCES

1. Horizontal diplopia is seen

2. Sixth nerve palsy is found in 10 to 20% of cases.
3. Transient Bell's type palsies of seventh cranial nerve rarely occur.
4. The cranial nerves which make nearly a 90 degree bend (CN 2, CN 6, CN 7) are more prone for damage at the site of the bend.

FALSE LOCALISING SIGNS

ABDUCENT NERVE PALSY

Raised intracranial pressure causes sixth nerve palsy. More susceptibility to damage is due to its sharp bend over the superior border of the petrous temporal bone and the downward shift of the brainstem towards foramen magnum produced by raised intracranial pressure.

PAPILLEDEMA

1. It is due to raised intracranial pressure.
2. It is the hallmark of Idiopathic intracranial hypertension[8].
3. It is the cardinal sign of IIH.
4. It may be bilateral, asymmetrical, or even unilateral.
5. It is the cause of visual loss in IIH patients.

PERIMETRY

1. Visual field defects occur in almost all cases of Idiopathic intracranial hypertension.
2. Most common visual field defects are
    a. Enlargement of blind spot.
    b. Inferior nasal step defect.
    c. Arcuate defects.
3. visual field loss may be progressive and severe
4. The earliest visual field defect-inferior nasal step
5. Blind spot enlargement is ubiquitous in IIH.

FEATURES OF FULMINANT IIH:

1. Rapid onset of symptoms
2. Significant visual loss
3. Macular edema
4. Cerebral venous thrombosis
5. It requires rapid treatment

MECHANISMS THAT LEAD TO DAMAGE OF THE OPTIC DISC ARE:

1. Disruption of axonal transport
2. Intraneuronal optic nerve ischemia

DISRUPTION OF AXONAL TRANSPORT

- -Raised cerebrospinal fluid pressure disturbs the normal gradient between intraocular and retrolaminar pressure.
- This leads to raised optic nerve tissue pressure.
- This finally results in axoplasmic stasis
- This results in intra-axonal edema.

INTRANEURONAL OPTIC NERVE ISCHEMIA

- High CSF pressure is transmitted to the region posterior to the optic nerve sheath.
- This increases the optic nerve tissue pressure
- This disturbs the pressure gradient across the lamina cribrosa.
- This results in axoplasmic flow stasis and compression of small arterioles.
- This finally results in intraneuronal ischemic damage to the optic nerve.

CAUSES OF LOSS OF VISUAL ACUITY IN IIH[7]

1. Chronic (atrophic) papilledema.
2. Chorioretinal folds.
3. Macular edema or exudates
4. Infarction of the optic disc
5. Subretinal peripapillary hemorrhage extending through the fovea
6. Subretinal peripapillary neovascular membrane

RISK FACTORS FOR VISUAL LOSS[7]

1. Recent weight gain
2. High grade papilledema
3. Atrophic papilledema
4. Subretinal hemorrhage
5. Significant visual field loss at presentation
6. hypertension

EVALUATION AND INVESTIGATIONS

1.NEUROIMAGING

Normal neuroimaging is a essential for the accurate diagnosis of IIH.

MAGNETIC RESONANCE IMAGING:

MRI with or without contrast is the best investigation of choice.

a. MRI Angiography

b. MRI Venography

- To rule out arterial disease and venous obstruction(thrombosis)
- Arnold-Chiari malformations
- To see structured lesions(mainly posterior fossa lesions)
- To see hydrocephalus
- Fascial resolution is better in MRI which provides three- dimensional image.
- Soft tissue lesions are well appreciated
- Magnetic resonance venogram is the procedure of choice for diagnosis of dural venous sinus thrombosis.
- On neuroimaging few patients show evidence of stenosis of one or both transverse sinuses.

COMPUTED TOMOGRAPHY

a. To rule out intracranial lesions that would produce increased intracranial pressure and rule out obstructive hydrocephalus.
   - Acute vascular causes- like subarachnoid, epidural, subdural, intracranial haemorrhages, acute infarctions.
   - After head injury- cerebral edema

b. Patient with contraindication to MRI like pacemaker, metallic clip and metallic foreign body.

2. LUMBAR PUNCTURE

a. Diagnostic- to evaluate for intracranial hypertension by recording the opening pressure.
b. To send CSF for microbial/infectious studies like-Total leukocyte, differential count, glucose, protein, cytology and VDRL.
c. Therapeutic-pseudotumor cerebri.

   It is usually contraindicated because of the danger of herniation of the brain into the foramen magnum which causes pressure on medulla leading to sudden death in cases with intracranial space occupying lesions with midline shift.

COMPLICATIONS OF LUMBAR PUNCTURE ARE:

1. Poor compliance
2. Painful
3. Difficult in obese patients
4. May produce a remission of Pseudotumor cerebri by creating a permanent fistula through the duramater.
5. Spinal epidermoid tumors
6. Infection

   A CSF pressure more than 250mm H2O is consistent with the diagnosis.

   The upper limit of CSF pressure in children is generally considered to be 180-200 mmH2O.

3. VISUAL FIELDS TESTING BY PERIMETRY

- Quantitative perimetry with kinetic perimetry for the peripheral field.
- Automated static perimetry for central fields.

MONITORING OF THE OPTIC NERVE HEAD

- Fundus photography is performed at the first evaluation and then whenever there is a change to provide the examiner with objective evidence of the appearance of the optic disc.
- Confocal scanning tomography is a new method to quantify the degree of papilledema.

5. CONTRAST SENSITIVITY

- It is a sensitive technique to record the optic nerve dysfunction.

6.VISUAL EVOKED POTENTIALS

- It is a useful tool but not routinely employed.

7.ULTRASONOGRAPHY

- It can be used to assess the diameter of the retrobulbar optic nerves as a measure of intracranial pressure.

X-RAY FINDINGS IN PAPILLEDEMA:

IN ADULTS:

a. Demineralisation of subcortical bone leading to loss of “lamina dura” (white line) of sellar floor followed by thinning of dorsum sella and the posterior clinoid process.
b. In extreme cases the sella becomes very shallow and flattened with its floor and anterior wall demineralised and the posterior clinoid process and dorsum sella destroyed.
c. Increased intracranial tension causes enlargement of the emissary veins in occipital region.
d. Congenital cyst or chronic subdural hematoma may show localised thinning or bulge.

X-RAY FINDINGS IN CHILDREN:

a. Presents with sutural diastasis, (sutural widening)
b. Increased convolutional markings with thinning of the bone.
c. Any separation beyond 2 mm is suspicious of increased tension.
d. Silver beaten appearance-due to pressure of sulci and gyri.

FUNDUS FLUORESCEIN ANGIOGRAPHY FINDINGS OF PAPILLEDEMA:

EARLY PHASE:

- Disc capillary dilatation
- Dye leakage spots
- Microaneurysms over the disc

LATE PHASE:

- Leakage of dye beyond disc margin
- Pooling of dye around the disc as vertically oval pooling

MRI FINDINGS IN IIH

1. Slit like ventricles
2. Optic nerve findings
    a. Prominence of subarachnoid space around the optic nerve
    b. Papilledema
    c. Flattening of the posterior sclera
    d. Vertical tortuosity of the optic nerves
    e. Enhancement of prelaminar optic nerves
3. Partial empty sella turcica

4. Small meningoceles (region of geniculate ganglion and orbital apex)
5. Prominent arachnoid pits (lateral sphenoid)
6. Transverse sinus stenosis is seen in MRA.

EMPTY SELLA

a. It is associated with prolonged rise in IOP
b. It is due to downward herniation of arachnocele through the diaphragm sellae.

POSTERIOR GLOBE FLATTENING

a. This is seen in both CT and MRI studies
b. It is the sine qua non neuroimaging sign of Pseudotumor cerebri
c. This is related to both raised intracranial as well as intraocular pressure.

CSF STUDIES:

To find the opening pressure and to rule out infective and neoplastic causes of Papilledema.

HUMPHREY FIELD ANALYSER

- It is the most frequently used automated perimeter[5].

- It is a type of static perimetry in which the location of a stimulus remains fixed at a certain location within the field, with the intensity increased until it is seen by the subject.
- The target intensity is increased until threshold is reached.

METHOD:

HFA consists of a hemispherical bowl onto which a target can be projected at any location in the visual field.

1. Monitor on the side of the instrument presents a series of menus. Background luminance is set at 31.5 asb, considered to be at the lower end of the photopic illumination range.
2. Variation in stimulus intensity can be achieved by altering target size or luminance. Size is set prior to the test; 4 sq.mm is used routinely.
3. Luminance is altered between 0.08 asb and 10 000 asb brighter than the background: between 51 dB and 0 Db.

a. In 30-2, the area tested extends to 30 degrees temporally as well as nasally.

RELIABILITY INDICES:

These reflect the extent to which patient's results are reliable. They should be analysed first. In patients who consistently fail to achieve good reliability indices, they can be switched over to a suprathreshold strategy or kinetic perimetry.

1. **Fixation losses:**

   They indicate steadiness of gaze during the test. A gaze monitor is used in newer HFAs.

2. **False positives:**

   These are detected in when a stimulus is accompanied by a sound. The grey scale printout appears pale.

3. **False negatives:**

   These are detected by presenting a stimulus much (9 dB) brighter than threshold at a location where the threshold has already been determined. If the patient fails to respond a false negative is recorded. A high false negative score indicates inattention or tiredness. The grey scale printout in individuals with high false negative responses tends to have a clover-leaf shape.

4. Interpretation:

With SITA strategies, false negatives or false positives about 15% should be regarded as highly significant. With full-threshold strategies, fixation losses over 20% and false positives or negatives over 33%.

HUMPHREY'S FIELD ANALYSER

MANAGEMENT OF IDIOPATHIC INTRACRANIAL HYPERTENSION

A. Treatment depends on the course of idiopathic intracranial hypertension.

B. When IIH is associated with an identified causal triggering factor, such as anemia, excessive vitamin A, Tetracyclines, obstructive sleep apnoea, withdrawal of the offending drug or treatment of the triggering factor often results in rapid improvement.

C. Main goals of the treatment are:

a. To preserve vision
b. To alleviate symptoms of raised intracranial pressure especially headache[3].

D. Management is requires history, examination and clinical course.

E. The most important factor is the amount of vision loss and the severity of patient's symptoms that disrupts their activities of daily living.

MEDICAL MANAGEMENT

It is aimed at reducing the intracranial pressure and relieving the patient of the distressing symptoms.

WEIGHT LOSS

Weight loss is a crucial in the treatment of overweight and obese patients with IIH[3]. A modest degree of weight loss is required for improvement in symptoms and signs (5% to 10% of total body weight). Low sodium diet and mild restriction of fluids is useful in some patients.

LUMBAR PUNCTURE

The treatment of IIH begins with the diagnostic lumbar puncture. The opening CSF pressure should be measured in the relaxed, lateral decubitus position without sedation to avoid spurious elevations of intracranial pressure. Smooth walled venous sinus stenosis resolve with lowering the CSF pressure. LP needle creates a sieve that allows sufficient regress egress of CSF, so that ICT is normalised.

INDICATIONS

DIAGNOSTIC

1. Infectitious
2. Inflammatory
3. Neoplastic
4. Meningitis
5. Subarachnoid hemorrhage

THERAPEUTIC

1. Hydrocephalus
2. Idiopathic intracranial hemorrhage
3. Spinal anaesthesia
4. Chemotherapy

CONTRAINDICATIONS

1. Uncal herniation
2. Skin infection at the puncture site
3. Sepsis

CORTICOSTEROIDS

Their use remains unclear in IIH. There is usually a recurrence of disc edema with rapid tapering of the dose. Use of long-term steroids is abandoned. They can be used for short-term treatment preoperatively before a CSF shunting procedure. Prednisolone is started at a dose of 2mg/kg/day in those who are intolerant to high doses of acetazolamide. This is given for two weeks and weaned over the next two weeks. Blood pressure, electrolytes, and urine glucose are monitored regularly.

ACETAZOLAMIDE

It is the most common drug used in the treatment of IIH. It is a carbonic anhydrase inhibitor. It decreases production of CSF at the level of the choroid plexus. It is usually started at a dose of 0.5 to 1 g/day and gradually increased until clinical improvement is seen. It also causes anorexia aiding in weight loss.

TOPIRAMATE

Topiramate is an alternative drug for IIH. It is primarily an anti-convulsant. It is a partial carbonic anhydrase inhibitor. It is effective for headaches such as migraine. It has weight loss as a prominent side effect.

FUROSEMIDE

It is a second line treatment. It is a loop diuretic. It lowers the intracranial pressure by both diuretic effect and reducing transport of sodium into the brain. Potassium supplementation is given as needed. It is initiated at a dose of 20mg BD and gradually increased to a maximum of 40mg TID.

SURGICAL THERAPY

These are mainly for treating refractory IIH.

CSF DIVERSION TECHNIQUES

1. Lumboperitoneal shunting
2. Ventriculoperitoneal shunting

NON-CSF DIVERSION TECHNIQUES

1. Optic nerve sheath fenestration
2. Induced weight loss by bariatric surgery
3. Venous sinus stenting

CSF shunting procedures:

They are mainly useful in cases of

a. Failed medical treatment
b. Medically intractable headache

The problems associated with this procedure are

a. Mechanical shunt dysfunction
b. Infections
c. Need for repeated revisions
d. High failure rate

The stereotactic ventriculoperitoneal shunting technique is recently introduced method. It is minimally invasive method.

OPTIC NERVE SHEATH DECOMPRESSION

It consists of creating a fenestration in the dural sheath just behind the globe. Exact mechanism is uncertain. It may be due to CSF egress forming a fistula thereby preventing transmission of high CSF pressure to the optic nerve head.

Loss of vision is a serious complication of idiopathic intracranial hypertension. Outcomes are to improve or stabilise the visual function. Optic nerve sheath decompression is an effective and safe surgical procedure to improve vision in patients with IIH. Optic nerve sheath decompression helps in relieving headache and improving visual acuity.

INDICATIONS FOR DECOMPRESSION

1. Failure of medical treatment as evidenced by clinical signs such as
    - Marked degree of swelling (>5D)
    - Great engorgement of veins
    - Presence of extensive hemorrhage
    - Early appearance of exudates spots

2. Progressive headaches unrelieved with medical treatment

3. Progressive optic neuropathy evidenced by early contraction of visual field.

4. Severe or rapid loss of vision at the onset, relative afferent papillary defect or signs of advanced optic nerve dysfunction.

5. Severe papilledema causing macular edema or exudates.

APPROACHES

1. Medial transconjunctival orbitotomy
2. Lateral orbitotomy
3. Lateral canthotomy

PROCEDURE

In this procedure, a window or multiple sites are made in the dural sheath of the optic nerve immediately behind the globe. This immediately reduces pressure on the nerve by creating a filtration apparatus that controls the pressure surrounding the orbital segment of the optic nerve.

MECHANISM

1. The filtering effect with local cerebrospinal fluid pressure reduction improving the peripapillary circulation.

2. Generalised decrease in the intracranial pressure after optic nerve sheath decompression.
3. The scarring of the arachnoid that may protect the optic nerve head from elevated cerebrospinal fluid pressure.

COMPLICATIONS OF ONSD

1. Extraocular motility restriction-commonly involves lateral rectus.
2. Pupillary dysfunction.
3. Loss of vision from vascular occlusion
4. Visual field defects
5. Orbital hemorrhage
6. Transient or protracted blindness
7. Globe perforation

OTHER DISC DECOMPRESSION TECHNIQUES TO RELIEVE PAPILLEDEMA ARE:

1. Subtemporal decompression
2. Suboccipital craniectomy

BARIATRIC SURGERY

It is a gastric exclusion procedure. It essentially treats comorbid conditions in obesity such as arterial hypertension, diabetes mellitus, and sleep apnoea. A neuro-endocrine basis is proposed for the benefits of this procedure. It produces a durable remission in IIH.

VENOUS SINUS STENTING

Neuroimaging shows narrowing or stenosis of transverse venous sinuses in a large number of IIH patients. Collapse of the transverse sinus is ubiquitous in IIH and obstructs venous return. This stenting procedure reduces cerebral venous pressure, reduces intracranial pressure, and improves the symptoms and signs. There is a persistent debate as to whether these venous abnormalities are cause or consequence of increased intracranial pressure.

MANAGEMENT OF PREGNANCY INDUCED HYPERTENSION WITH PAPILLEDEMA

1. Bed rest
2. Diet restriction
3. Sedatives like phenobarbitone/diazepam
4. Control of blood pressure

5. Control edema- proteinuria/diuretic/hypertonic glucose
6. Finally if patient does not respond to treatment, pregnancy has to be terminated.

TREATMENT OF FULMINANT IIH

1. intravenous corticosteroids
2. insertion of lumbar drain

DETERMINATION OF THERAPEUTIC SUCCESS

1. relief of headache
2. diminished frequency of transient visual obscuration
3. regression of papilledema
4. stability or improvement of field defects
5. weight reduction

OVERALL MANAGEMENT PROTOCOL

1. No symptoms of papilledema
    - periodic monthly review
    - if vision normal for 3 months, then 2-monthly review
2. With transient obscuration of vision/signs of optic nerve dysfunction

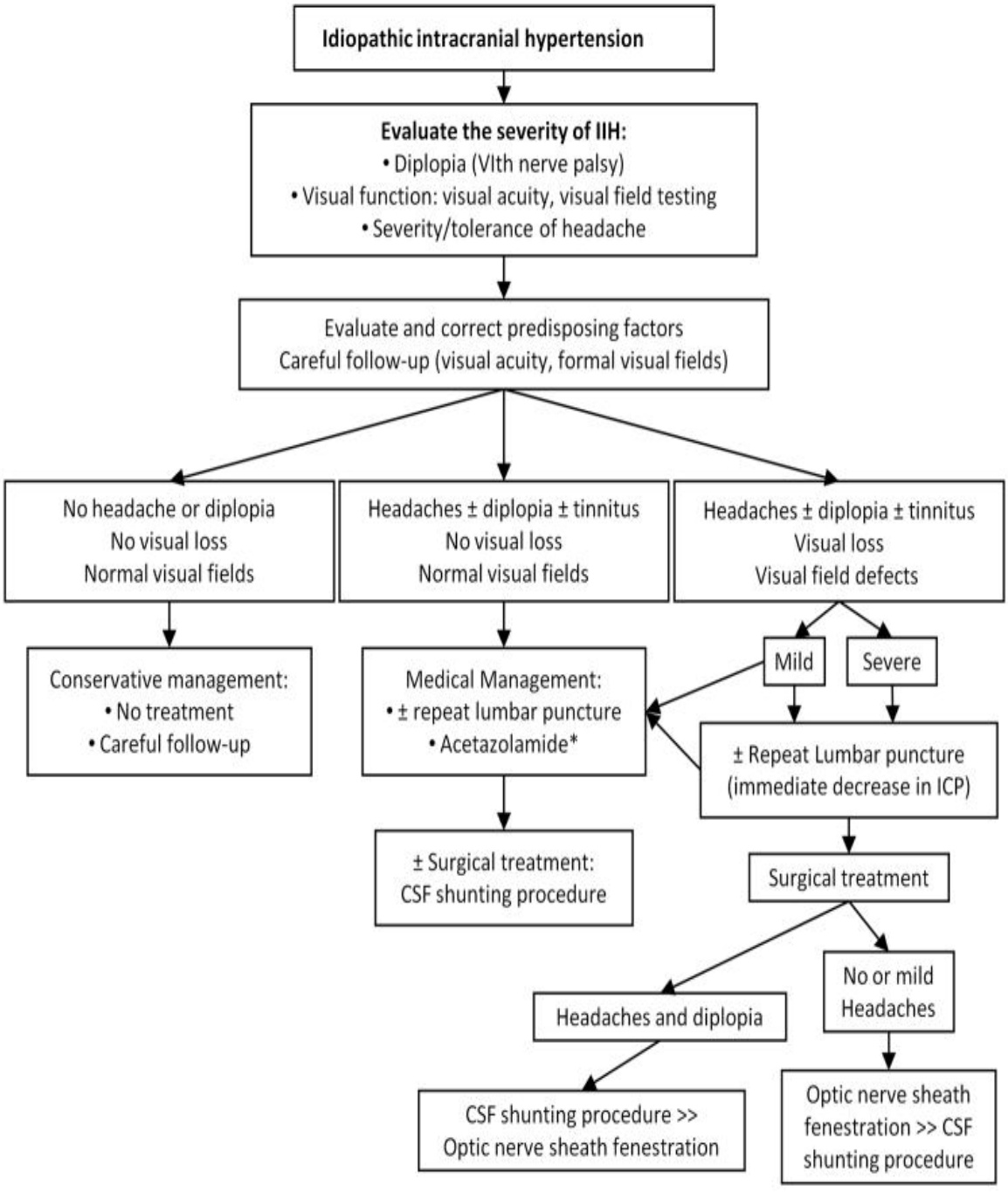
- Acetazolamide 1g daily depending on patient's tolerance
- Re-examine the patient every 2 to 3 weeks for signs of compromise

3. With progressive optic neuropathy

    - Acetazolamide- corticosteroid (80 to 100 mg/day)

4. Other treatment modalities reserved for refractory cases

    - Repeated lumbar puncture
    - Lumboperitoneal shunt
    - Optic nerve sheath decompression
    - Venous sinus stenting



In all patients who are overweight, weight loss is essential; bariatric surgery sometimes recommended in morbid obesity

ICP: intracranial pressure

*Although there has been no clinical trial evaluating treatment of IIH, acetazolamide is commonly prescribed (between 1 and 2 grams per day) to decrease CSF production

REVIEW OF LITERATURE

Sandeep Randhawa and Gregory P.Van Stavern studied

current standard of care in the diagnosis and treatment of idiopathic intracranial hypertension and concluded "that newer treatment modalities are being explored for IIH refractory to standard medical therapy, but their efficacy and safety must be demonstrated in large studies before they can be adopted as part of standard treatment"[8].

Martin G.Radvany et al described " visual and neurological outcomes following Endovascular Stenting for Pseudotumor Cerebri associated with Transverse sinus stenosis[9]".

D Soler et al described that " the correct diagnosis of Benign Intracranial hypertension relies on the recognition of the typical symptoms, radiological exclusion of a mass lesion, and recognition of the possible diagnostic pitfalls"[10].

Ambika S et al described "the clinical profile, evaluation and management of idiopathic intracranial hypertension"[1].

Honorat R et al concluded that " visual prognosis is generally better in children than in adults and no risk factors for visual sequelae were identified[11]".

Hannerz J and Ericson K studied " The relationship between idiopathic intracranial hypertension and obesity"[12].

Wall M et al described the risk factors for poor visual outcome in patients with idiopathic intracranial hypertension[13].

Obi EE et al described that " Optic nerve sheath fenestration is a safe procedure and predominantly stabilises visual function in majority of maximally medicated patients[14"].

Supuran CT studied and concluded that "Acetazolamide is effective in the treatment of Idiopathic intracranial hypertension[15"].

Mulla Y et al studied and concluded that " Quality of life in IIH patients is significantly reduced and effective headache management is required to improve quality of life in IIH patients[16"].

Kanagalingam S and **Subramanian PS** described the "role of Cerebral venous stenting in the management of patients with refractory pseudotumor cerebri[17"].

Elder BD et al described that venous stenting represents a viable treatment option for fulminant idiopathic intracranial hypertension[18].

Naarden MT described that patients with IIH are mainly overweight young women who present with raised intracranial pressure[19].

Ibrahim YA et al described the presence of Transverse dural venous attenuation on CT scans in 96% of patients with IIH and concluded that Venous attenuation sign is an additional imaging marker in the evaluation of IIH patients[20].

Wardly DE described " Obstructive sleep apnoea as a potential risk factor for the development of Idiopathic intracranial hypertension"[21].

Dave SB and Subramanian PS studied the treatment options for Pseudotumor cerebri and concluded "that weight reduction and medical management may be utilised for IIH cases without vision loss and surgical procedures for patients with severe vision loss".[22]

Banta JT and Farris BK described that "Optic nerve sheath decompression is a safe and effective means of stabilising visual acuity in patients with Pseudotumor cerebri with progressive visual loss despite maximal medical therapy".[23]

Hingwala DR et al studied the imaging signs in patients with Idiopathic intracranial hypertension and concluded that Optic nerve head protrusion and globe flattening were associated with IIH[24].

Fridley J et al described that bariatric surgery may be an effective treatment for IIH in obese patients, both in terms of symptom resolution and visual outcome.[25]

Valerie Biousse, Beau B. Bruce and Nancy J Newman studied the pathophysiology and management of IIH. They described that " Vitamin A metabolism, adipose tissue and cerebral venous abnormalities are associated with IIH"[26].

Harvey J Sugerman et al described that gastric bariatric surgery was associated with resolution of Pseudotumor cerebri in morbidly obese women[27].

Susan P Mollan et al studied and concluded that " IIH requires a multidisciplinary approach including ophthalmologists, neurologists and neurosurgeons. They also suggested that disease modifying therapy is weight loss and CSF shunting to be considered temporary".[28]

John Chen described that "IIH is a disease of women in child-bearing years, and its prevalence is increasing due to the worldwide obesity epidemic"[29].

N.N.Baheti et al studied the long-term visual outcomes of IIH and concluded that "IIH patients can have delayed worsening or relapses and about one-tenth of patients have permanent visual loss early or late in the course of the disease"[30].

Bryan D.Riggeal et al studied the clinical course of IIH with Transverse sinus stenosis and described that transverse sinus stenosis is common in IIH and concluded that there is no correlation between the degree of Transverse sinus stenosis and the clinical course[31].

J.Alexander Fraser et al studied the risk factors for IIH in men and suggested a possible role of sex hormones and obstructive sleep apnoea in the pathogenesis of IIH in men[32].

AIMS AND OBJECTIVES

- To study the clinical profile of patients with idiopathic intracranial hypertension.
- To study the behaviour and the natural course of idiopathic intracranial hypertension.
- To study the neuroimaging features and to look for hypoplasia and stenoses of transverse and sigmoid sinuses.
- To analyse the response to treatment and study the visual outcome.

MATERIALS AND METHODS

STUDY DESIGN

Hospital based prospective study

SOURCE OF DATA

Neuro ophthalmic services, Aravind Eye Hospitals

STUDY SUBJECTS

Patients who were proven to have idiopathic intracranial hypertension both clinically and radiologically from December 2014 to May 2015 (6 months) were enrolled for study.

SAMPLE SIZE

61 Patients who were proven to have idiopathic intracranial hypertension clinically and radiologically from a period of December 2014 to May 2015 for a period of 6 months who presented to the Department of Neuroophthalmology, Aravind Eye Hospital, Madurai.

STUDY PERIOD

December 2014 to May 2015(6 months)

DATA ANALYSIS

STATISTICAL METHODS

Mean (SD) or Frequency (Percentage) was used to describe summary information. Statistical analysis was done by STATA 11.1 (Texas, USA).

INCLUSION CRITERIA

It includes all patients with any of the following symptoms and signs with radiologically proven idiopathic intracranial hypertension

- Signs and symptoms of idiopathic intracranial hypertension such as headache, transient obscuration of vision, defective vision, vomiting, neck pain, giddiness, field defects and diplopia.
- No localising focal neurological signs except unilateral or bilateral sixth nerve paresis.
- Normal neuroimaging results adequate to exclude cerebral venous thrombosis and intracranial tumours i.e magnetic resonance imaging of the brain, often with additional sequences (computed tomography or magnetic resonance venography)
- Reproducible visual field defects.

EXCLUSION CRITERIA

- Abnormalities on neurological examination aside from papilledema and its related visual loss or sixth nerve paresis.
- Abnormal neuroimaging except for an empty sella, thickening of optic nerve sheath complex, stenoses of transverse and sigmoid sinuses and widening of peri-optic subarachnoid space.

CLINICAL EVALUATION

A series of 61 patients who presented to our Neuro ophthalmology department with clinically and radiologically proven diagnosis of idiopathic intracranial hypertension were included in our study. All these patients underwent a thorough ophthalmological and neurological evaluation.

The patients' particulars like name, age, sex, address were documented in a proforma specially designed for the study, and was filled by the examining doctor.

A detailed history of each and every symptom of the patient was taken such as the onset, duration, progression, associated factors, aggravating and relieving factors were documented.

The patients were also enquired about the past medical and surgical history, systemic illness, treatment history, personal history and family history.

EACH ONE OF THE PATIENT INCLUDED IN OUR STUDY HAS TO UNDERGO ROUTINE OPHTHALMIC EVALUATION

- Visual acuity by Snellen's chart
- Refraction
- Pupillary reaction for normal pupil, sluggish pupil or RAPD.
- General ophthalmic examination with torch light and slit lamp biomicroscopy
- Intraocular pressure measurement by non-contact tonometry
- Fundus examination by direct ophthalmoscope and slit lamp biomicroscopy using 90 Dioptre lens and indirect ophthalmoscopy.
- Extraocular movement examination using torch light
- Colour vision evaluated by Pseudo-isochromatic Ishiharas chart
- Central fields by Bjerrums screen
- Visual fields by Automated perimetry (HFA 30-2)
- A complete neurological evaluation was done to every patient including general consciousness, cranial nerve examination, motor system evaluation and sensory system evaluation were done.

- Neuroimaging was done in all patients either CT Brain or MRI/MRV with or without contrast depending upon the need and affordability of the individual patients.
- During follow up visual acuity, pupil reaction, colour vision, fields and fundus examination were done at one month and three months.
- Vitals such as pulse rate and blood pressure were recorded
- Weight, height and body mass index were done
- Systemic examination of cardiovascular, respiratory, central nervous system were done.
- Investigations such as haemoglobin percentage, random blood sugar, lipid profile and thyroid profile were done.
- Fundus photographs were taken for every patient
- During follow up weight, visual acuity, pupillary reaction, colour vision, central fields and fundus was done at one month and three months.

ANALYSIS AND RESULTS

Analysis of collected data was done based on the following

1. Incidence
2. Age and sex distribution
3. Presence or absence of various symptoms
    a. Yes-1
    b. No-2
4. Duration of symptoms
    a. <1 month
    b. 1 month to 1 year
    c. > 1 year
5. Associated clinical features
    a. Yes-1
    b. No-2
6. Presence of systemic illness
    a. Yes-1
    b. No-2

7. Past treatment history
    a. Yes-1
    b. No-2
8. Personal history
    a. Yes-1
    b. No-2
9. Body mass index
        a. Healthy 18.5 to 25 -1
        b. Overweight 25 to 30 -2
        c. Obese 30 to 35 -3
        d. Severely obese >35 -4
10. Best corrected visual acuity
        a. 6/6 to 6/60 -1
        b. 5/60 to 1/60 -2
        c. <1/60 -3
11. Intraocular pressure by non-contact tonometry
12. Anterior segment examination
13. Pupil examination
        a. Normal
        b. Sluggish

c. RAPD

14. Colour vision by Ishihara's chart

    a. Normal

    b. Defective

15. Central fields by Bjerrums screen

    a. Normal

    b. Defective

16. Fundus examination

    a. Early papilledema

    b. Established papilledema

    c. Chronic papilledema

    d. Resolving papilledema

    e. Optic atrophy

17. Investigations-

    a. Random blood sugar

    b. Haemoglobin

    c. Fasting lipid profile

    d. Thyroid function test

18. Radiological findings
    a. Computed tomography
    b. Magnetic resonance imaging of brain
    c. Magnetic resonance venogram
19. Fundus photography
20. Visual fields by automated perimetry using Humphrey's Field Analyser(30-2)
21. Treatment-Medical or Surgical
22. Visual acuity at follow up- Static/Improved/Worsened
23. Fundus status at follow up

AGE

Mean (SD) of the age is 29.97(8.41) years and it ranges from 16 years to 49 years

GENDER

Gender	n	%
Male	5	8.2
Female	56	91.8
Total	61	100

DEFECTIVE VISION

Defective vision	n	%
Yes	18	29.5
No	43	70.5
Total	61	100

DURATION OF DEFECTIVE VISION

Duration of defective vision	**n**	**%**
<1month	11	61.1
1month – 1 year	7	38.9
Total	18	100

Complaints	**n(%)**
Headache	54(88.5)
Transient Obscuration of Vision	25(41.0)
Field defects	1(1.6)
vomiting	23(37.7)
Diplopia	9(14.8)
Neck pain	14(22.9)
Giddiness	5(8.2)

SYSTEMIC DISEASE

Systemic Disease	n	%
Diabetes/ Hypertension/ Thyroid	1	1.6
Hypertension	3	4.9
Hypertension/ Dyslipidemia/ PCOD	1	1.6
Hypothyroid	3	4.9
Pregnancy induced Hypertension/ PCOD	1	1.6
Nil	52	85.3
Total	61	100

TREATMENT HISTORY

Treatment History	n	%
Anti-DM/ Anti-HT/ Thyroxine	1	1.6
Anti-HT	3	4.9
Anti-HT/ OCP	1	1.6
Doxy/ Vit A	1	1.6
OCP	1	1.6
Thyroxine	3	4.9
Nil	51	83.6
Total	61	100

VITALS

Vitals	Mean(SD)	Min – Max
Weight	67.59(12.98)	47 – 100
Height	151.98(6.16)	140 – 177
IOP	14.25(2.27)	10 – 20
Blood Pressure		
Systolic BP	119.90(15.73)	90 – 160
Diastolic BP	77.77(11.48)	50 - 120

WEIGHT

Weight	Mean(SD)	Min – Max
Visit1	67.59(12.98)	47 – 100
Visit2	66.35(12.55)	47 – 98
Visit3	66.44(12.91)	50 – 98

BODY MASS INDEX

BMI	n	%
Healthy	14	23.0
Overweight	29	47.5
Obese	11	18.0
Severely Obese	7	11.5
Total	61	100

OCULAR EXAMINATION

BCVA

BCVA(n=122)	Visit1	Visit2	Visit3	Final
6/6 – 6/60	121(99.18)	113(100.0)	86(100.0)	113(100.0)
5/60 – 1/60	-	-	-	-
<1/60	1(0.8)	-	-	-

PUPIL

Pupil(n=122)	Visit1	Visit2	Visit3	Final
Normal	104(85.2)	106(93.0)	86(100.0)	110(96.5)
Sluggish	16(13.1)	7(6.1)	-	3(2.6)
RAPD	2(1.6)	1(0.9)	-	1(0.9)

EOM

EOM	n	%
Normal	113	92.6
Abnormal (6^{th} nerve paresis	9	7.4
Total	122	100

FUNDUS

Fundus(n=122)	Visit1	Visit2	Visit3	Final
Early Papilledema	52(42.6)	4(3.5)	-	-
Established Papilledema	66(54.1)	24(21.1)	2(2.3)	4(3.5)
Chronic Papilledema	4(3.3)	5(4.4)	2(2.3)	4(3.5)
Chronic papilledema and Optic atrophy	-	2(1.8)	-	2(1.7)
Normal	-	10(8.8)	46(53.5)	56(49.1)
Resolving papilledema	-	69(60.5)	36(41.9)	48(42.1)

COLOR VISION

Color Vision	Visit1	Visit2	Visit3	Final
Normal	115(94.3)	110(98.2)	74(100.0)	110(98.2)
Defective	7(5.7)	2(1.8)	-	2(1.8)

CENTRAL FIELDS

Central Fields	Visit1	Visit2	Visit3	Final
Normal	104(85.2)	106(94.6)	71(97.3)	108(96.4)
Defective	18(14.8)	6(5.4)	2(2.7)	4(3.6)

INVESTIGATIONS

Investigations	Normal	Abnormal
Hemoglobin	34(55.7)	27(44.3)
RBS	61(100)	-
Lipid profile	56(93.3)	4(6.7)
TFT	55(90.2)	6(9.8)
HFA 30-2	30(49.2)	31(50.8)

CT BRAIN

CT Brain	n	%
1	8	66.7
1 and 2	4	33.3
Total	12	100

MRI

MRI	n	%
1	19	35.9
1 and 2	34	64.1
Total	53	100

CT Brain/ MRI	n	%
CT Brain	8	13.1
MRI	53	86.9
Total	61	100

CT / MRI - Final	n	%
1	23	37.7
1 and 2	38	62.3
Total	61	100

MRV

MRV	n	%
B/L TSS	1	1.9
HLTS/ HLSS	16	30.2
HLTS/ HLSS/ RTS/ RSS	2	3.8
HRTS/ HRSS/ LTS	1	1.9
LTS	1	1.9
LTS/ LSS	1	1.9
RTS	1	1.9
Normal	30	56.6
Total	53	100

TREATMENT

Treatment	Visit1(n=61)	Visit2(n=55)	Visit3(n=34)	Final
DMX	13(21.3)	12(21.8)	5(14.7)	11(20.0)
DMX, IS	5(8.2)	2(3.6)	1(2.9)	1(1.8)
DMX, WR	24(39.3)	23(41.8)	7(20.6)	16(29.1)
DMX, WR, IS	15(24.6)	11(20.0)	9(26.5)	12(21.8)
LP	1(1.6)	-	-	-
MANNITOL	1(1.6)	1(1.8)	-	-
ONSD	2(3.3)	-	-	-
TOPIRAL	-	2(3.6)	1(2.9)	1(1.8)
TOPIRAL, WR	-	1(1.8)	-	-
WR	-	2(3.6)	8(23.5)	10(18.2)
WR, IS	-	1(1.8)	1(2.9)	2(3.6)
IS	-	-	2(5.9)	2(3.6)

DISCUSSION

Idiopathic intracranial hypertension can present with varied clinical symptoms, neuro-ophthalmic features and radiological features. It is a disorder of elevated cerebrospinal fluid pressure of unknown cause that usually occurs in obese women in the childbearing years.

In our prospective study we included 61 patients. The mean age group is 29.97 years which ranges between 16 years to 49 years. This mean age is younger than that recorded as 32.89 years in a study conducted by Ambika S et al in a tertiary referral ophthalmic centre in India[1] and almost equal to the age in another study which was conducted in North America in which it was 29.0 years[2]

In our study, out of 61 patients 56(91.8%) were females and 5(8.2%) were males which is almost equal to the retrospective study conducted in a tertiary ophthalmic centre in India[1]**. John Chen** and **Michael Wall** found that female gender is an obvious risk factor for IIH since 90% of the affected population were obese females.[29]

The most common complaints in our study patients were headache(88.5%), transient obscuration of vision(41.0%), vomiting(37.7%),

defective vision(29.5%), neck pain(22.9%), diplopia(14.8%), giddiness(8.2%) and field defect(1.6%). The most common complaint is headache(88.5%) in our study group which is more than the study conducted in India[1] which was 76%. Most of our patients presented within one month of duration.

Among our study patients, 9 patients (14.7%) had systemic illness history such diabetes, hypertension, dyslipidemia, pregnancy induced hypertension and polycystic ovarian disease. Remaining 52 patients (85.3%) had no systemic illness**. Inbal Avisor Et Al** reported the association of PCOD and IIH.[33]

In our study, 2 patients gave history of treatment with oral contraceptive pills for menstrual irregularities, 1 patient took treatment for acne with oral doxycycline and vitamin A capsules and 3 patients had treatment history with thyroxine for hypothyroidism. **John Chen and Michael Wall** in a study reported that vitamin A intoxication and other forms of Vitamin A such as Isotretinoin given for the treatment of acne are also associated with IIH[29]

The body mass index was calculated as weight in kilograms divided by height in meters squared in our study and 47.5% were overweight, 23% were healthy, 18% were obese and 11.5% were severely obese. **Daniels AB Et Al** found that higher BMIs were associated with greater risk of IIH.[5] **Aimee J.**

Szewka Et Al did a retrospective study and reported that higher BMI at diagnosis is associated with increased severe visual loss in patients with IIH.[35]

On presentation, Best corrected visual acuity was found to range from 6/6 to Hand movements in our study population. Out of 122 eyes, 121 eyes had visual acuity between 6/6 to 6/60 and one eye had visual acuity of hand movements on presentation. In a retrospective study in India[1] , 55 eyes had visual acuity of 6/6, 20 eyes had visual acuity between 6/9 to 6/18, 7 eyes had visual acuity between 6/24 to 6/60 and 18 eyes had <6/60.

In our study, Pupillary examination was normal in 104 eyes(85.2%), sluggish in 16 eyes(13.1%) and Relative Afferent Pupillary defect in 2 eyes(1.6%).

Extraocular movements were normal in 92.6% and abnormal in 7.4%. Most common involvement in our study group was the sixth nerve. Horizontal diplopia was reported due to sixth nerve paresis which is a false localising sign in IIH. In a study by **Michael Wall**, he reported horizontal diplopia in 1/3rd of IIH patients and sixth nerve paresis were found in 10 to 20% of cases.[29]

Fundus examination of our patients showed early papilledema in 52 eyes(42.6%), established papilledema in 66 eyes(54.1%) and chronic

papilledema in 4 eyes(3.3%). One patient progressed to optic atrophy as a result of chronic papilledema. Papilledema is the most common sign and is usually symmetric, but can be highly asymmetric or unilateral in 10% of patients[36]. In our study, papilledema was bilateral in 59 patients but asymmetric in 2 patients with one eye early papilledema and one eye established papilledema.

In our study, Colour vision was defective in 7 eyes(5.7%) and normal in 115 eyes(94.3%). In retrospective study in India, colour vision analysis showed that colour vision was normal in 16 patients and affected in 10 patients.[1]

Central fields were normal in 104 patients(85.2%) and defective in 18 patients(14.8%) in our study population. Most common defect was an enlarged blind spot in the affected patients.

In our study, blood investigations such as haemoglobin, random blood sugar, fasting lipid profile and fasting thyroid profile were done for all the patients. Haemoglobin values were normal in 34 patients(55.7%) and below normal in 27 patients(44.3%). Random blood sugar values were normal in all the patients(100%). Lipid profile was normal in 56 patients(93.3%) and abnormal in 4 patients(6.7%). Thyroid profile was normal in 55 patients(90.2%) and altered in 6 patients(9.8%).

Our study highlights the possible association between iron deficiency anemia and IIH evidenced by resolution of papilledema after correction of iron deficiency. **Bhavna Et Al** studied iron deficiency masquerading as idiopathic intracranial hypertension in a 13 year old girl.[37]

In our study, 6 patients were hypothyroid and 4 were on thyroxine replacement therapy. **Ester Coutinho Et Al** reported Grave's disease as a rare cause of Pseudotumor cerebri[38]. Thyroid disturbances in the form of hypothyroidism, hyperthyroidism and thyroxine replacement therapy have unique correlation with this disorder.

Automated perimetry was done by Humphrey's Field analysis 30-2 to assess visual field loss in our study. Fields were normal in 30 patients(49.2%) and abnormal in 31 patients(50.8%). Most common defects were enlarged blind spot and generalised constriction of fields. In a retrospective study conducted in India, the most common visual field defect was an enlarged blind spot which was noted in 60 eyes and other field defects were advanced generalised constricted fields as well as nasal and arcuate defects in seven eyes.[1] In a prospective study conducted by **Michael Wall,** the most common defects were enlargement of physiologic blind spot and loss of inferonasal portions of the visual field along with constriction of isopters.[13]

Neuroimaging was done in all our patients (Computed tomography of brain and Magnetic Resonance Imaging with Magnetic Resonance Venography). Patients who were not affordable for MRI and MRV underwent imaging by Computed tomography. 53 patients underwent neuroimaging by MRI and MRV. Eight Patients underwent neuroimaging by CT.

In neuroimaging, 38 Patients showed both thickening of optic nerve sheath with widening of perioptic subarachnoid space and partial empty sella. 23 patients showed only thickening of optic nerve sheath with widening of perioptic subarachnoid space alone.

Out of the 53 patients who underwent MRI and MRV, 23 patients had either congenital hypoplasia or stenosis of transverse sinus or sigmoid sinus or both. In the other 30 patients, MRV was normal without stenoses or hypoplasia. There was no evidence of venous thrombosis in any of our patients ruling out cerebral venous thrombosis.

Out of our 23 patients, 16 (30.2%) had hypoplasia of left transverse sinus stenosis and hypoplasia of left sigmoid sinus stenosis, 2 patients had hypoplasia of both left transverse and left sigmoid sinus stenoses with right transverse sinus and right sigmoid sinus stenoses, 1patient had bilateral transverse sinus stenoses, 1 patient had hypoplasia of both right transverse and

right sigmoid sinuses with left transverse sinus stenosis, 1 patient had left transverse sinus stenosis alone, 1 patient had right transverse sinus stenosis alone and 1 patient had both left transverse and sigmoid sinus stenoses.

In a retrospective study done in 20 patients with Pseudotumor cerebri by **Brodsky MC** and **Vaphiades M**, the MRI disclosed flattening of posterior sclera in 80%, empty sella in 70%, distension of the perioptic subarachnoid space in 45%, enhancement of the prelaminar optic nerve in 50%, vertical tortuosity of the orbital optic nerve in 40% and intraocular protrusion of the prelaminar optic nerve in 30%[39]

M Neil Et Al studied the presence of bilateral transverse sinus stenosis causing idiopathic intracranial hypertension in a 19 years old man and suggested that a defective sinus venous drainage is primarily responsible for IIH[40]

Out of the 61 patients, 24 with high BMI were treated with Diamox tablets (Acetozolamide 250mg BD) and were advised Weight reduction, 15 patients who had coexisting Anaemia and with high BMI were treated with Diamox tablets and iron supplements along with Weight reduction, 13 whose BMI was normal were treated with Diamox alone, 1 patient underwent lumbar puncture for both diagnostic and therapeutic purposes, one patient was initially

treated with intravenous Mannitol and maintained with Diamox in the subsequent follow ups and 2 patients underwent surgical decompression by unilateral Optic Nerve Sheath Decompression. Two patients developed intolerance to Diamox and were treated with Topiramate tablets (Topiral).

. Weight reduction should be recommended for all obese and overweight IIH patients. Weight control improves the overall quality of life. Carbonic anhydrase inhibitors like acetazolamide 1gm daily or 250mg QID and methazolamide are the main medical treatment classically prescribed for IIH. [26] They decrease CSF production by inhibiting carbonic anhydrase in the choroidal plexus and also by mild dieresis. Side effects include lethargy, paraesthesia and altered taste sensation.[22]

Topiramate is a sulfa-derivative monosaccharide which acts by blockage of voltage gated sodium channels. It is mainly licensed for treatment of epilepsy and migraine prophylaxis. It is also an anti-obesity agent.

Symptoms of raised ICT and papilledema improve with treatment of anaemia. The mechanism is mainly by increased cerebral blood flow that maintains cerebral oxygen delivery.[16]

Mannitol is the most commonly used hyperosmolar agent for the treatment of IIH.[16]It has both rheologic and osmotic effects. It causes reduction in hematocrit and in blood viscosity, thereby increasing cerebral blood flow and increase in oxygen delivery.[42]

Lumbar puncture is most commonly done for evaluation of an acute headache and to rule out inflammatory or infective conditions of nervous system. The treatment of raised Intracranial pressure itself begins with diagnostic lumbar puncture which is often effective in relieving the symptoms and signs.[26]

Optic nerve sheath fenestration is done mainly for visual symptoms. It has also been proven recently to improve headaches. It is an effective long-term procedure for visual loss in IIH.[43] In ONSF, the dural sheath surrounding the retrolaminar portion of the optic nerve is fenestrated which creates a fistula between the subarachnoid space and orbital cavity. Finally, there is a reduction in the pressure around the nerve leading to resolution of papilledema and improvement of visual symptoms.

Transverse sinus stenoses is a common finding in IIH. It has been proposed that external compression of the venous sinus due to raised ICP results in stenoses. Stenting of the stenoses may reduce cerebral venous

pressure[43] **Mohamed S Teleb Et Al** did a study on systematic analysis of Transverse sinus stenting in IIH.[44] Endovascular stenting for dural venous stenosis is a safe and feasible procedure.

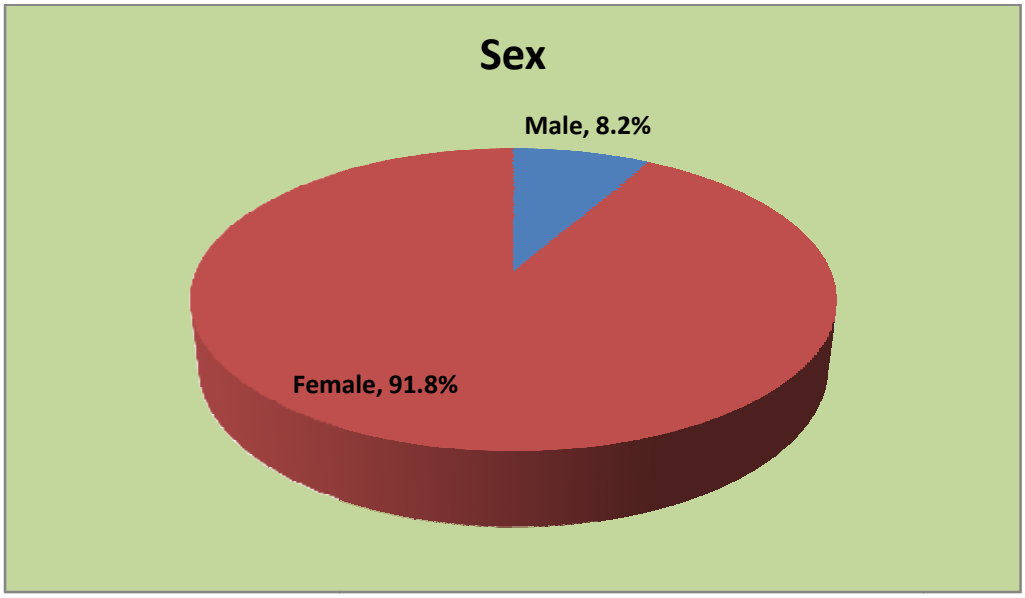
CONCLUSION

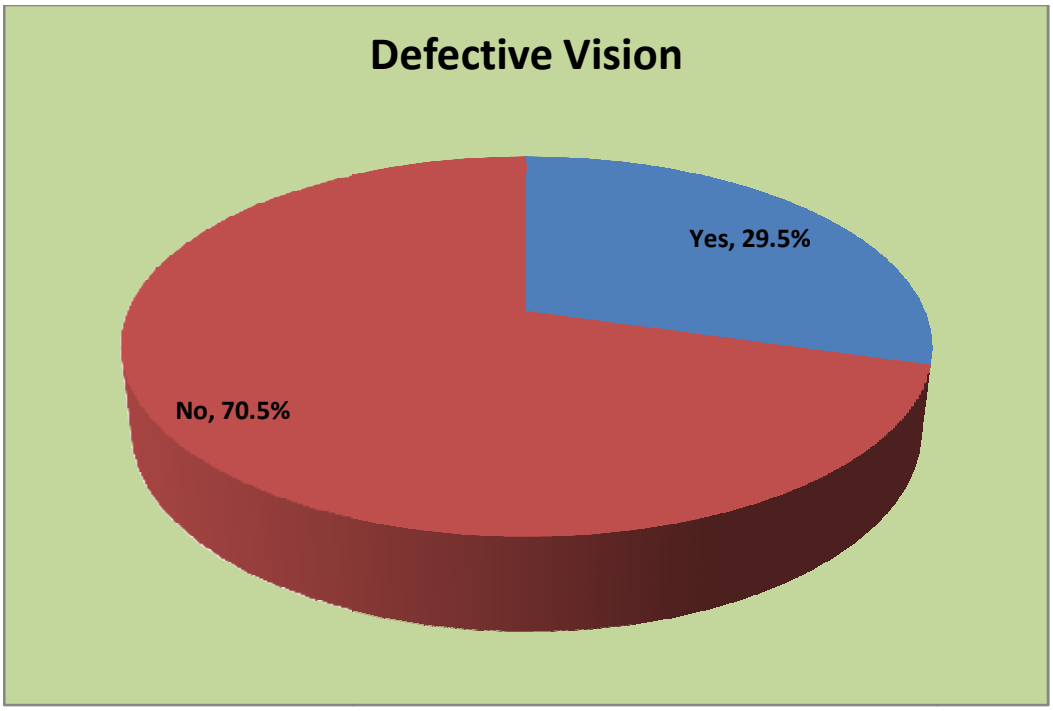
Idiopathic intracranial hypertension is characterized by elevated CSF pressure without apparent cause. Obese women of child-bearing age are more commonly involved than males. Most common complaint was headache in our study patients. Most of our patients presented within one month duration. Commonly associated risk factors are thyroid dysfunction, anemia, pregnancy, hypertension, exposure to exogenous drugs like tetracyclines, oral contraceptives, vitamin A supplements and thyroid replacement therapy. Anemia was a significant risk factor in our study population. High body mass index and recent weight gain is associated with IIH. Visual acuity was 6/6 in most eyes in our study. Sixth nerve paresis occurs as a false localizing sign in IIH. Most of the patients presented with established papilledema. Most common visual field defect is an enlarged blind spot in both Central fields test by Bjerrums Screen and Automated perimetry. In MRI and CT brain, thickening of optic nerve sheath and empty sella were the most common findings in our patients. In MRV study, 30.2%(16) patients had hypoplasia of left transverse sinus stenosis and hypoplasia of left sigmoid sinus stenosis. Patients who had high BMI were treated with Acetazolamide 250mg BD with Weight reduction. Patients who had coexisting Anaemia were treated with iron

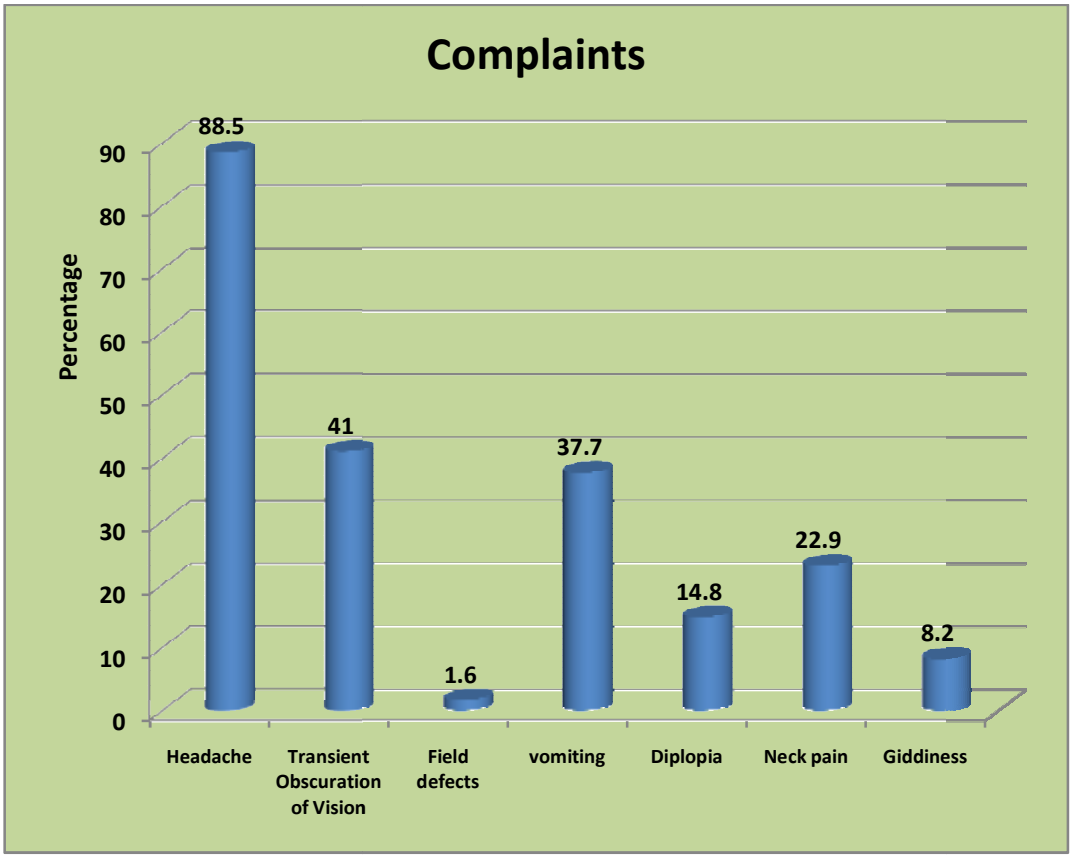
supplements along with acetazolamide. One patient was treated by lumbar puncture and two patients were treated with unilateral Optic Nerve Sheath Fenestration. Follow up at one month and at three months showed resolving papilledema in most cases.

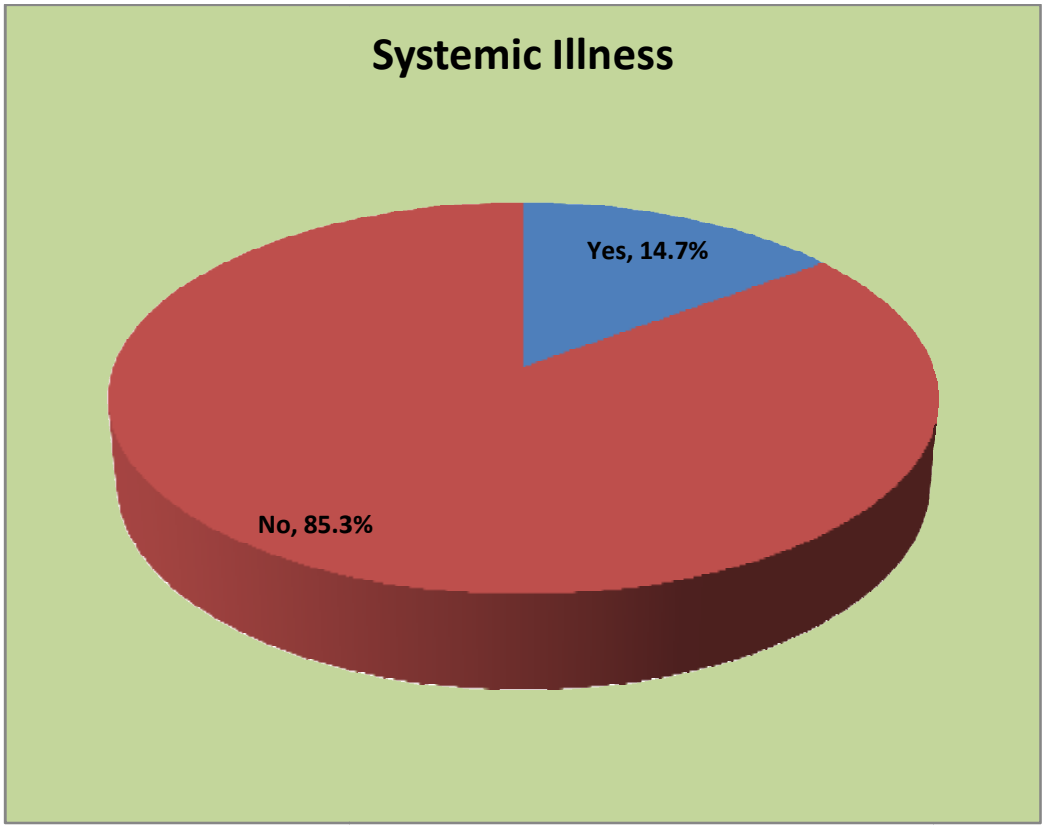
LIMITATIONS

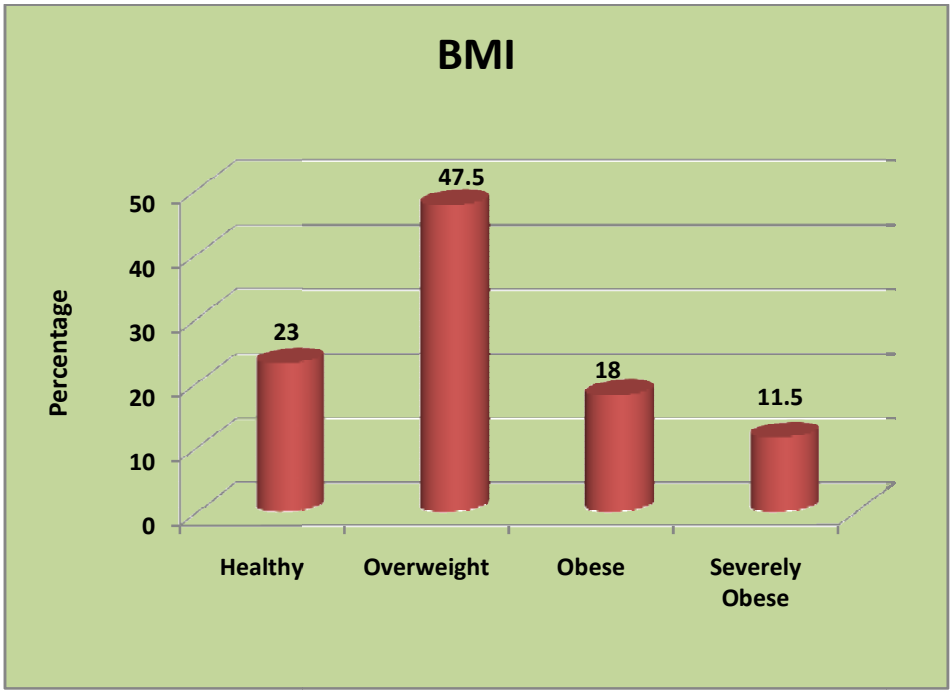
1. Few patients who were not affordable for MRI/MRV underwent CT study.
2. Few patients were lost for follow up.
3. Lack of a comparison group limiting our ability to assess the degree of stenosis typical of normal individuals.

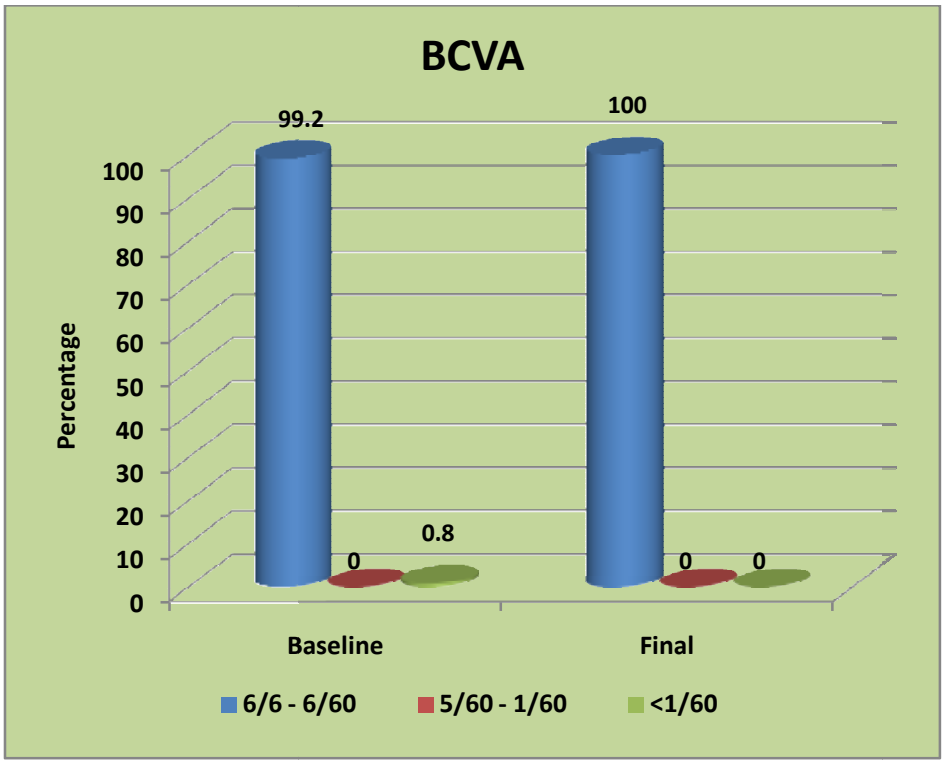

Sex
Male, 8.2%
Female, 91.8%

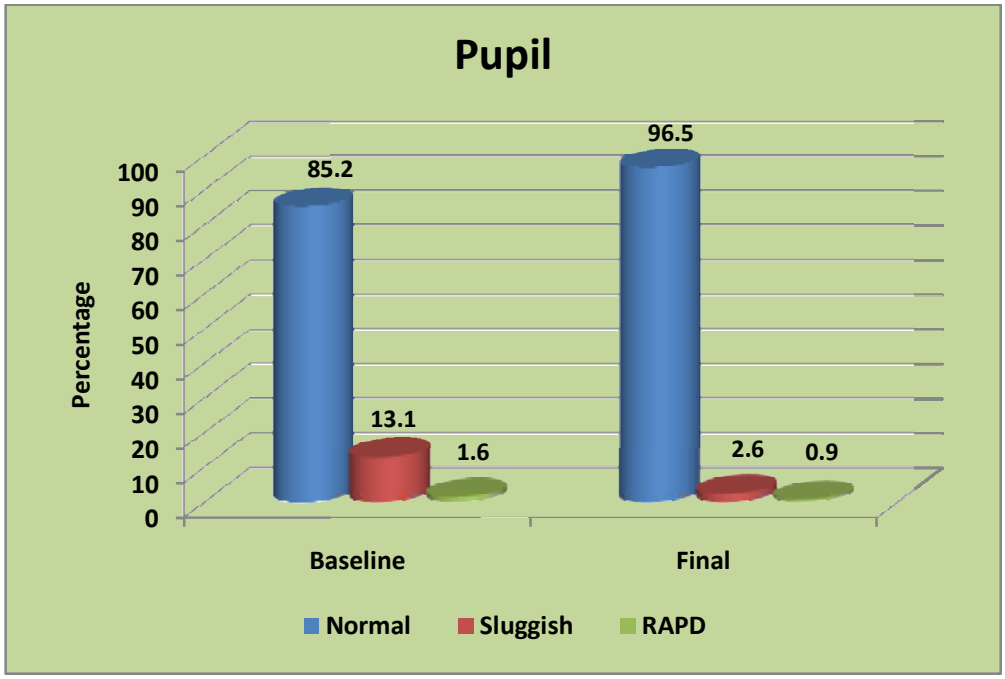

Defective Vision
Yes, 29.5%
No, 70.5%

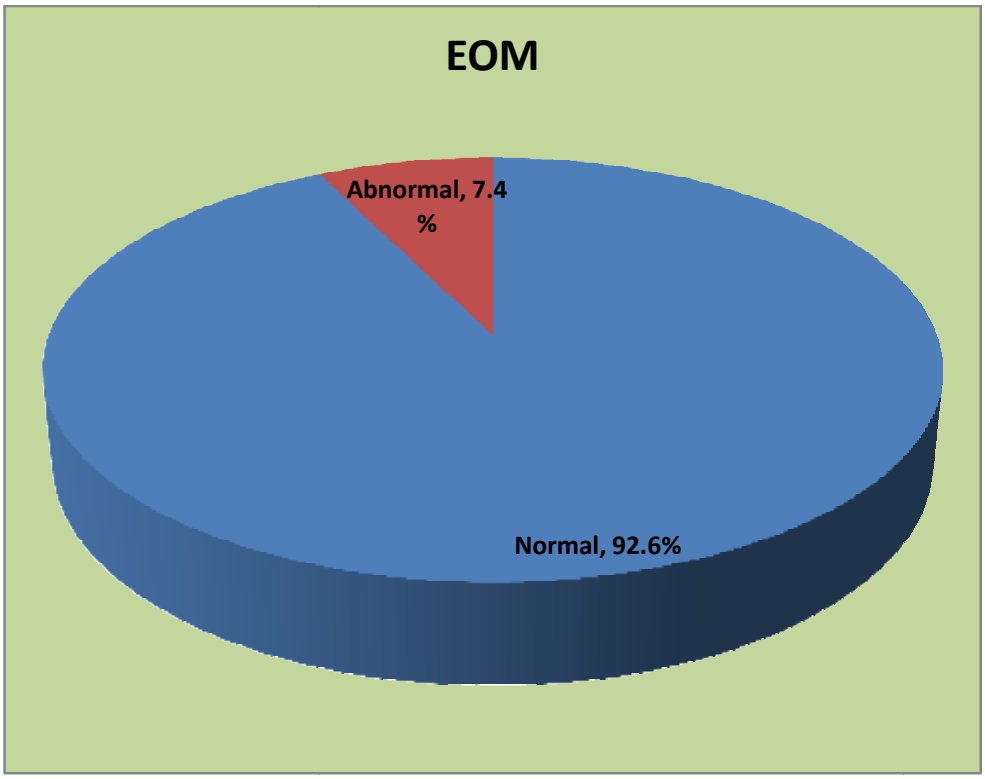

Complaints
Percentage
0
10
20
30
40
50
60
70
80
90
88.5
41
1.6
37.7
14.8
22.9
8.2
Headache
Transient Obscuration of Vision
Field defects
vomiting
Diplopia
Neck pain
Giddiness

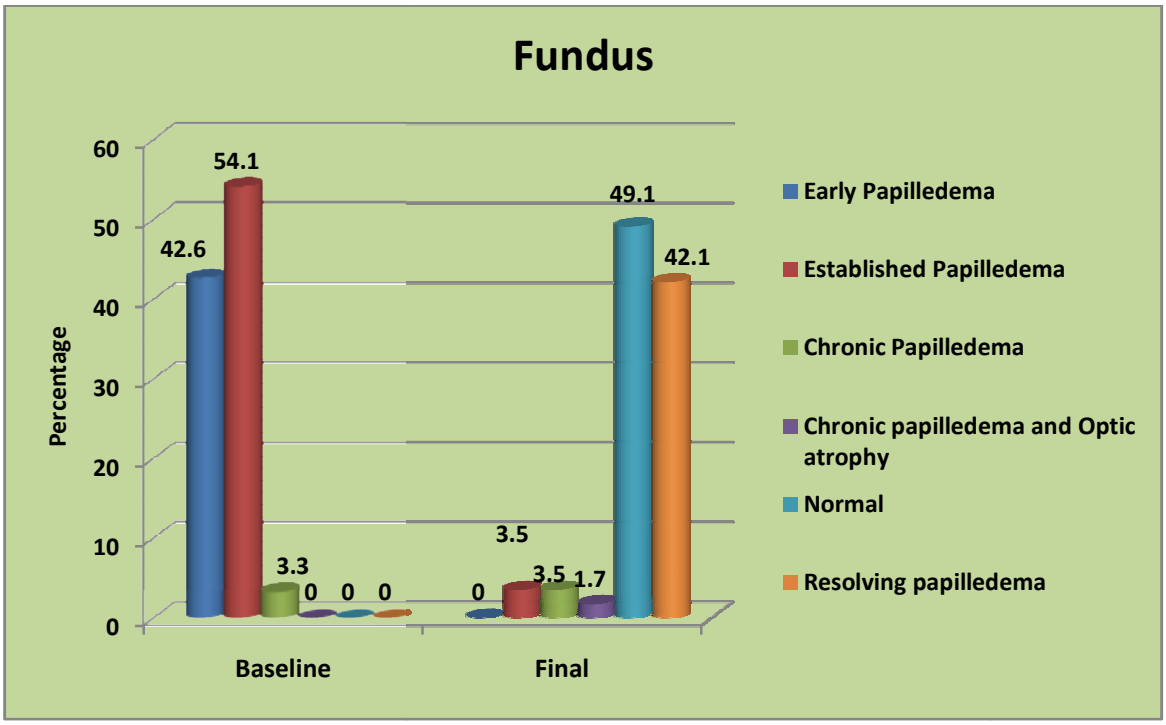

Systemic Illness
Yes, 14.7%
No, 85.3%

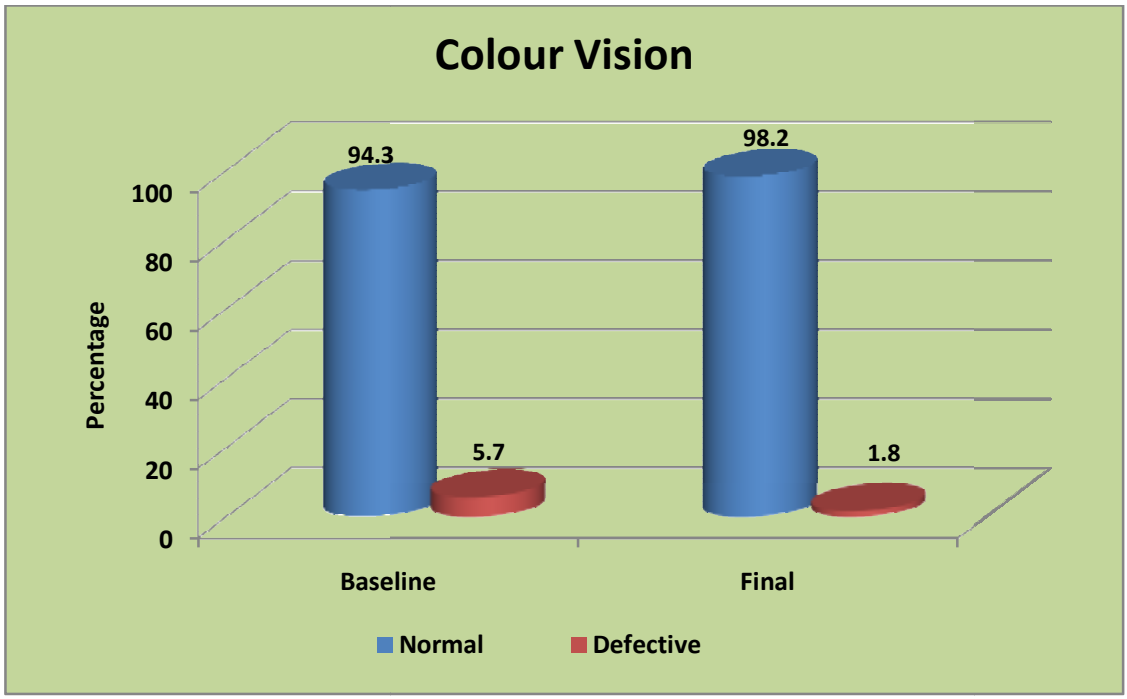

BMI
Percentage
50
40
30
20
10
0
47.5
23
18
11.5
Healthy
Overweight
Obese
Severely Obese

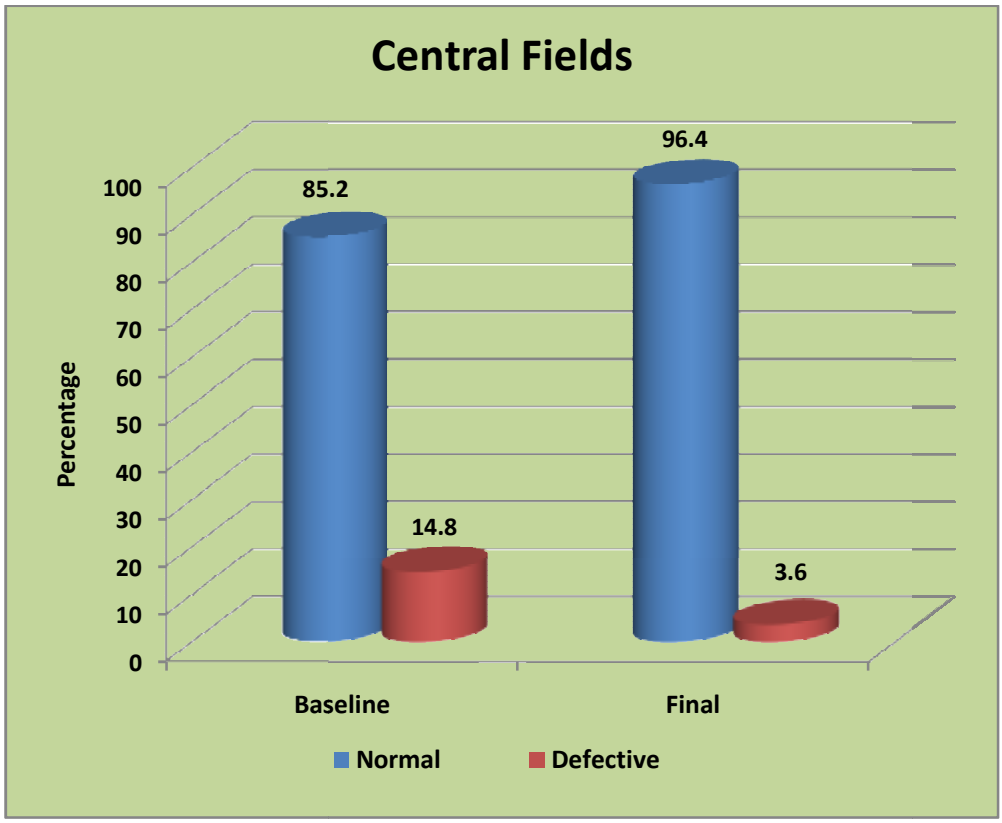

BCVA
Percentage
100
90
80
70
60
50
40
30
20
10
0
99.2
0
0.8
100
0
0
Baseline
Final
6/6 - 6/60
5/60 - 1/60
<1/60


Pupil
Percentage
100
90
80
70
60
50
40
30
20
10
0
85.2
13.1
1.6
96.5
2.6
0.9
Baseline
Final
Normal
Sluggish
RAPD

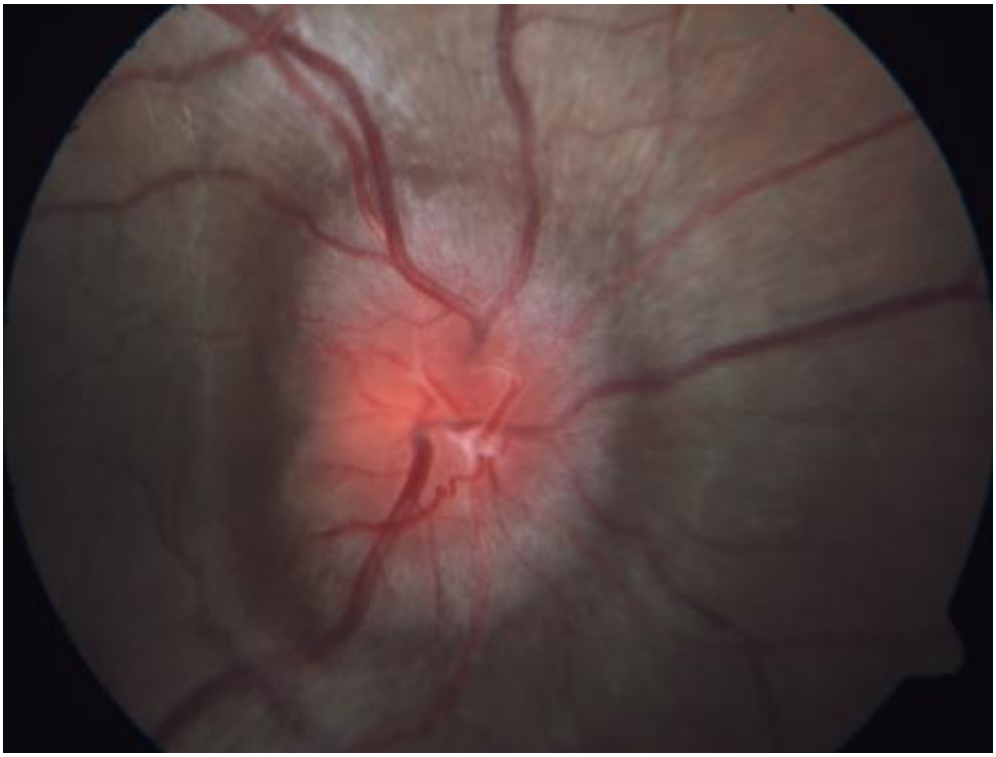

EOM
Abnormal, 7.4 %
Normal, 92.6%

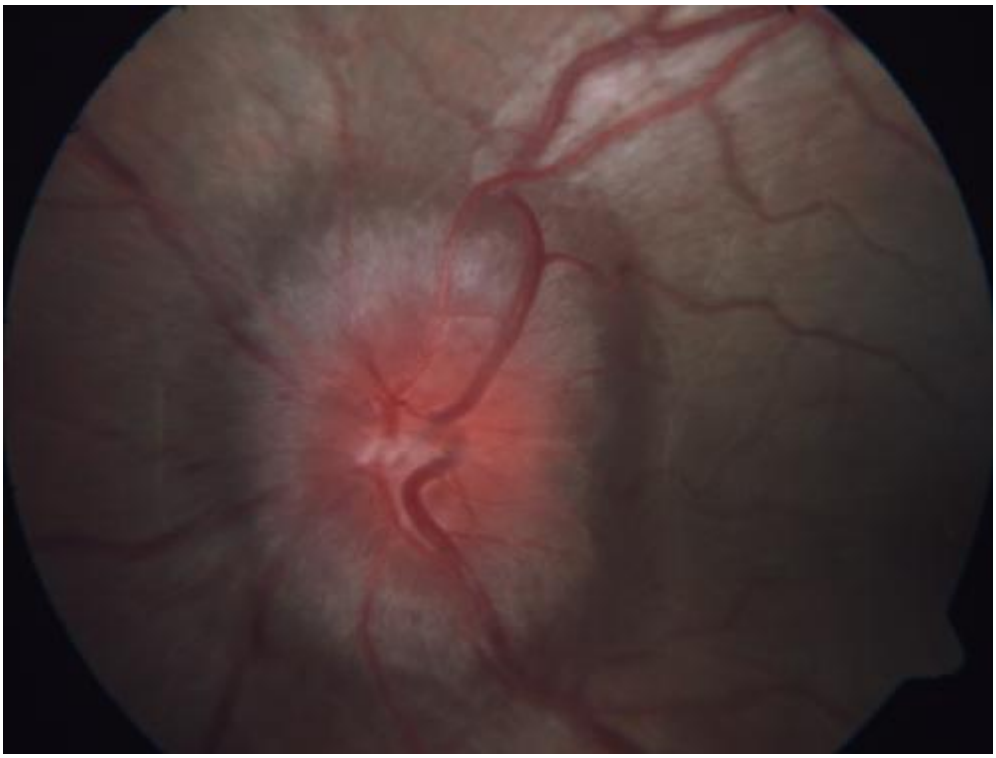

Fundus
Percentage
60
50
40
30
20
10
0
54.1
42.6
3.3
0
0
0
3.5
0
3.5
1.7
49.1
42.1
Baseline
Final
Early Papilledema
Established Papilledema
Chronic Papilledema
Chronic papilledema and Optic atrophy
Normal
Resolving papilledema


Colour Vision
Percentage
100
80
60
40
20
0
94.3
5.7
98.2
1.8
Baseline
Final
Normal
Defective

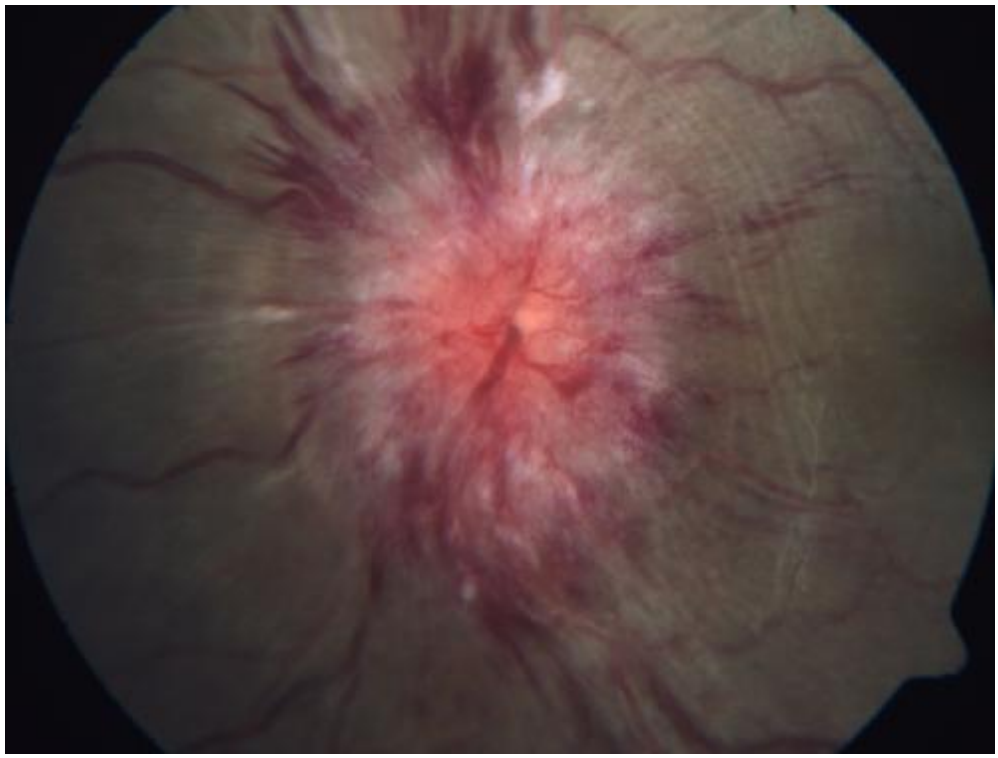

Central Fields
Percentage
100
90
80
70
60
50
40
30
20
10
0
85.2
14.8
96.4
3.6
Baseline
Final
Normal
Defective

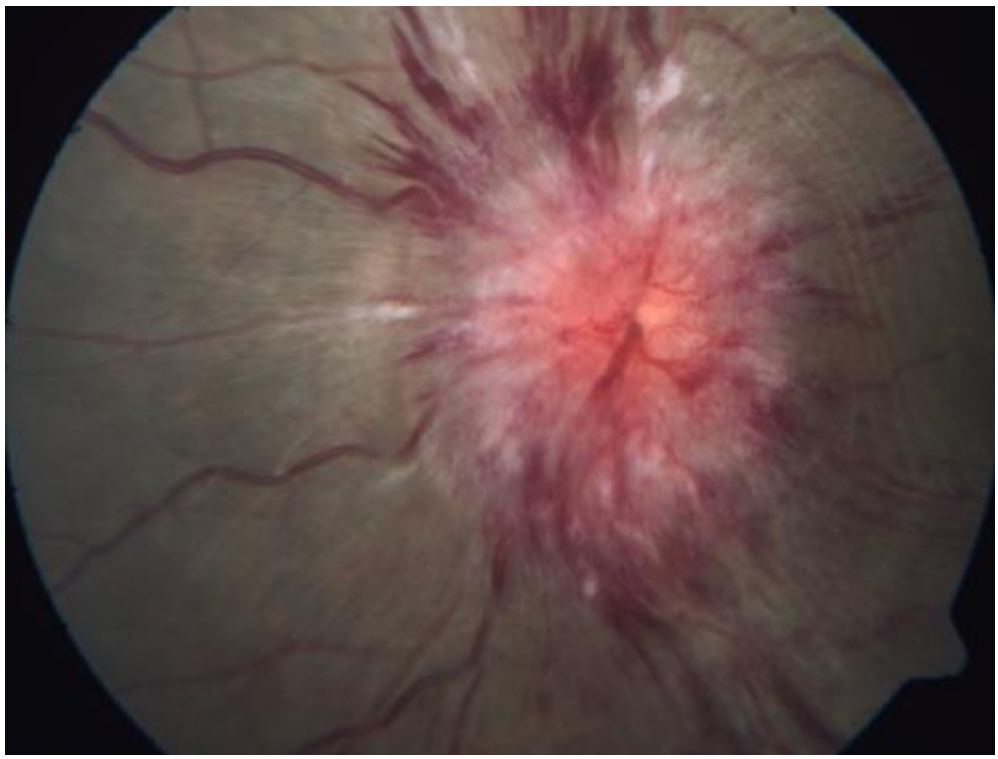
EARLY PAPILLEDEMA



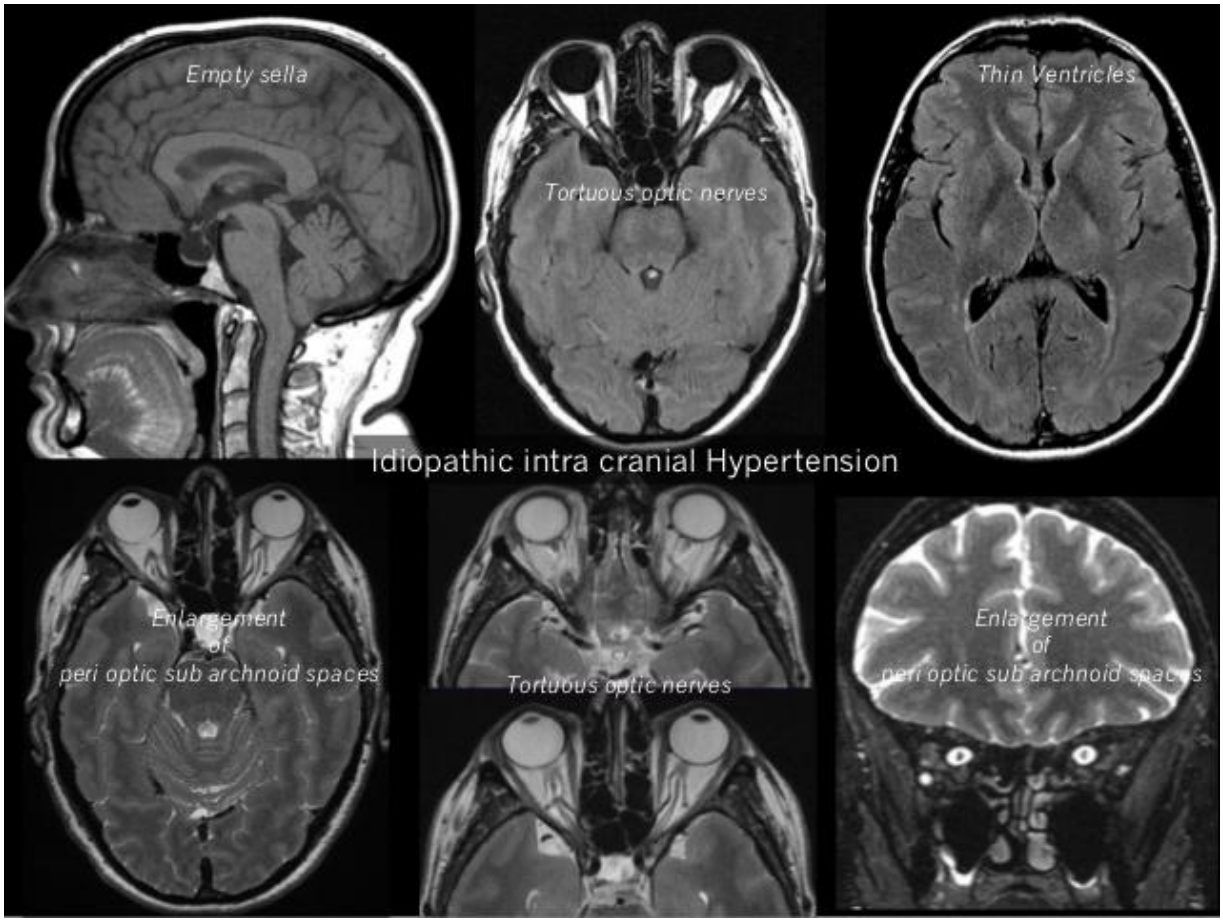


ESTABLISHED PAPILLEDEMA

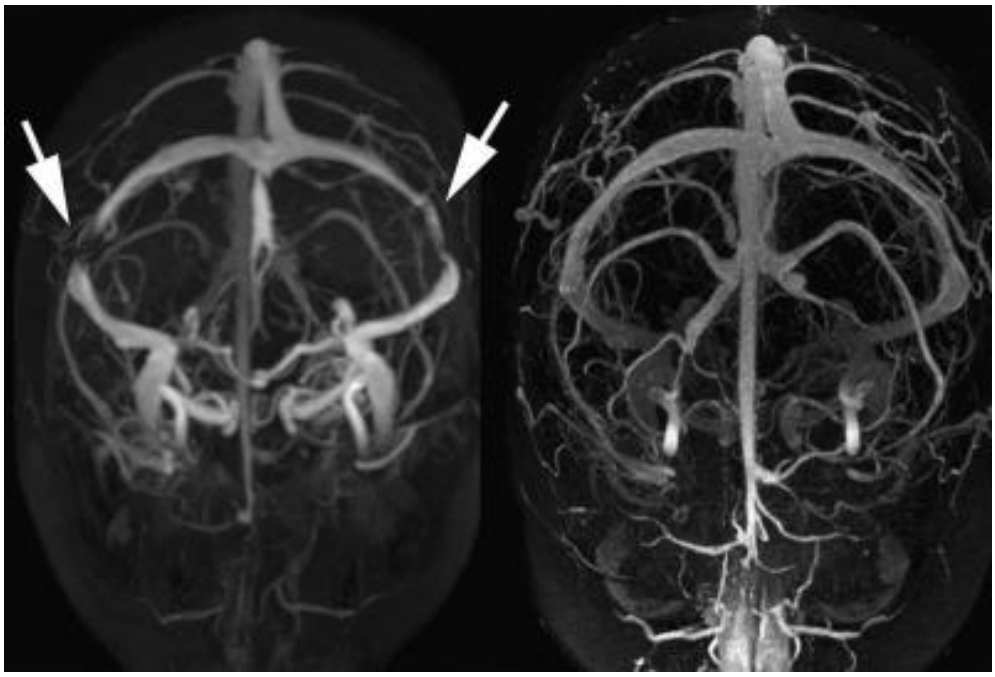




MRI FEATURES OF IIH



VENOUS STENOSIS



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PROFORMA-IDIOPATHIC INTRACRANIAL HYPERTENSION

CASE NO -

DATE

M.R.NO

NAME

AGE/SEX

OCCUPATION

PLACE

COMPLAINTS

HEADACHE -YES / NO

COMPLAINTS

TRANSIENT OBSCURATION OF VISION – YES / NO **1- YES**

DEFEECTIVE VISION - DURATION **2- NO**

- UNILATERAL / BILATERAL

DURATION

- SUDDEN / INSIDIOUS **1- < 1 MONTH**

- PAINFUL / PAINLESS **2- 1 MONTH TO 1 YEAR**

- PROGRESSIVE / STATIONARY **3- > 1 YEAR**

FIELD DEFECTS - YES / NO

VOMITING - YES / NO

DIPLOPIA - YES / NO

NECK PAIN - YES / NO

GIDDINESS - YES / NO

PAST HISTORY PAST H/O

DIABETES - YES / NO **1- YES 2- NO**

HYPERTENSION - YES / NO **DIABETES-DM**

DYSLIPIDEMIA - YES / NO **HYPERTENSION-HT**

CARDIAC ILLNESS - YES / NO **HYPOTHYROID**

BRONCHIAL ASTHMA- YES / NO **PCOD-POLYCYSTIC OVARIAN DISEASE**

MIGRAINE - YES / NO **DYSLIPIDEMIA-DYSLIP**

THYROID DISEASE - YES / NO **PIH-PREGNANCY INDUCED HYPERTENSION**

TREATMENT HISTORY TREATMENT H/O

ANTI-DIABETIC **1- YES 2- NO**

ANTI-HYPERTENSIVE

THYROID DRUGS

CARDIAC DRUGS

HYPERLIPIDEMIA

ORAL CONTRACEPTIVES (OCP)

VITAMIN A SUPPLEMENTS

PERSONAL HISTORY PERSONAL H/O

SMOKER	**- YES / NO**	**1- YES 2- NO**
ALCOHOLIC	**- YES / NO**	
SNORING	**- YES / NO**	
DIET	**- VEG / NON-VEG**	

FAMILY HISTORY FAMILY H/O

1- YES 2-NO

VITALS

PULSE RATE

BLOOD PRESSURE (BP)

BODY WEIGHT (KG)

HEIGHT (CM)

BODY MASS INDEX (BMI) **BMI= Weight (kg)/height^2(metres)**

CVS

RS

CNS

ABDOMEN

1-HEALTHY 18.5 TO 25

2-OVERWEIGHT 25 TO 30

3-OBESE 30 TO 35

4-SEVERELY OBESE >35

OCULAR EXAMINATION

RE **LE**

BCVA

BCVA

IOP **1-**

6/6 TO 6/60

ANTERIOR SEGMENT **2- 5/60 TO**

1/60

PUPIL **3- <1/60**

NORMAL / SLUGGISH / RAPD **PUPIL**

COLOUR VISION (CV) **1-**

NORMAL

CENTRAL FIELDS (CF) **2-**

SLUGGISH

FUNDUS **3- RAPD**

DISC EDEMA **CV and**

CF

BLURRING OF DISC MARGIN **1-**

NORMAL 2-DEFECTIVE

DISC HYPEREMIA **EP- EARLY**

PAPILLEDEMA

SPLINTER HEMORRHAGES **ESP-**

ESTABLISHED PAPILLEDEMA

MACULAR EDEMA **CP- CHRONIC**

PAPILLEDEMA

PATON'S LINES

DILATED TORTUOUS VESSELS

HARD EXUDATES

MACULAR FAN / STAR

BACKGROUND RETINA

OTHERS

INVESTIGATIONS

INVESTIGATIONS

HEMOGLOBIN

BLOOD GLUCOSE

LIPID PROFILE

THYROID PROFILE

CT SCAN

MRI AND MRV

FUNDUS PHOTO

HFA 30-2

TREATMENT

1- NORMAL

2-ABNORMAL

CT SCAN

1-THICKENING OF OPTIC NERVE SHEATH WITH WIDENING OF PERIOPTIC SUBARACHNOID SPACE

2- PARTIAL EMPTY SELLA

MRI

1- THICKENING OF OPTIC NERVE SHEATH WITH WIDENING OF PERIOPTIC SUBARACHNOID SPACE

2-PARTIAL EMPTY SELLA

MRV

HLTS- HYPOPLASIA OF LEFT TRANSVERSE SINUS

HLSS- HYPOPLASIA OF LEFT SIGMOID SINUS

LTS-LEFT TRANSVERSE SINUS STENOSIS

LSS- LEFT SIGMOID SINUS STENOSIS

RTS- RIGHT TRANSVERSE SINUS STENOSIS

RSS- RIGHT SIGMOID SINUS STENOSIS

HFA 30-2 **1- NORMAL** **2-ABNORMAL**

TREATMENT **DMX- DIAMOX**

WR- WEIGHT REDUCTION

IS- IRON SUPPLEMENTS

LP-LUMBAR PUNCTURE

ONSD- OPTIC NERVE SHEATH DECOMPRESSION

FOLLOW UP AT 1 MONTH

WEIGHT

HEIGHT

BMI

BLOOD PRESSURE

OCULAR EXAMINATION

RE

LE

BCVA

IOP

ANTERIOR SEGMENT

PUPIL

NORMAL / SLUGGISH / RAPD

COLOUR VISION

CENTRAL FIELDS

FUNDUS

DISC EDEMA

BLURRING OF DISC MARGINS

DISC HYPEREMIA

SPLINTER HEMORRHAGES

MACULAR EDEMA

PATON'S LINES

DILATED TORTUOUS VESSELS

HARD EXUDATES

MACULAR FAN / STAR

BACKGROUND RETINA

OTHERS

INVESTIGATIONS

FUNDUS PHOTO

FOLLOW UP AT 3 MONTHS

WEIGHT

HEIGHT

BMI

OCULAR EXAMINATION

RE **LE**

BCVA

IOP

ANTERIOR SEGMENT

PUPIL

NORMAL / SLUGGISH / RAPD

COLOUR VISION

CENTRAL FIELDS

FUNDUS

DISC EDEMA

BLURRING OF DISC MARGINS

DISC HYPEREMIA

SPLINTER HEMORRHAGES

MACULAR EDEMA

PATON'S LINES

DILATED TORTUOUS VESSELS

HARD EXUDATES

MACULAR FAN / STAR

BACKGROUND RETINA

OTHERS

INVESTIGATIONS

FUNDUS PHOTO

ABBREVIATIONS

BCVA	-	Best Corrected Visual Acuity
RAPD	-	Relative Afferent Pupillary Defect
CT	-	Computed Tomography
MRI	-	Magnetic Resonance Imaging
MRV	-	Magnetic Resonance Venography
ONSD	-	Optic Nerve Sheath Decompression
ICP	-	Intra Cranial Pressure
IIH	-	Idiopathic Intracranial Hypertension
BMI	-	Body Mass Index
CSF	-	Cerebrospinal Fluid
ONSF	-	Optic Nerve Sheath Fenestration
ONH	-	Optic Nerve Head
ICT	-	Intracranial Tension
MRA	-	Magnetic Resonance Angiography
IOP	-	Intraocular Pressure
HFA	-	Humphrey's Field Analyser

KEY TO MASTER CHART

DOV - Date of Visit

TOV - Transient Obscuration of Vision

EOM - Extraocular Movements

BCVA - Best Corrected Visual Acuity

IOP - Intraocular Pressure

BP - Blood pressure

V/A - Visual Acuity

CV - Colour Vision

CF - Central Fields

TFT - Thyroid Function Tests

FUNDUS

EP - Early Papilledema

ESP - Established Papilledema

CP - Chronic Papilledema

RP - Resolving Papilledema

OA - Optic Atrophy

N - Normal

CT AND MRI/MRV

LTS - Left Transverse Sinus Stenosis

RTS - Right Transverse Sinus Stenosis

LSS - Left Sigmoid Sinus Stenosis

HLTS - Hypoplasia of Left Transverse Sinus

HLSS - Hypoplasia of Left Sigmoid Sinus

HRSS - Hypoplasia of Right Sigmoid Sinus

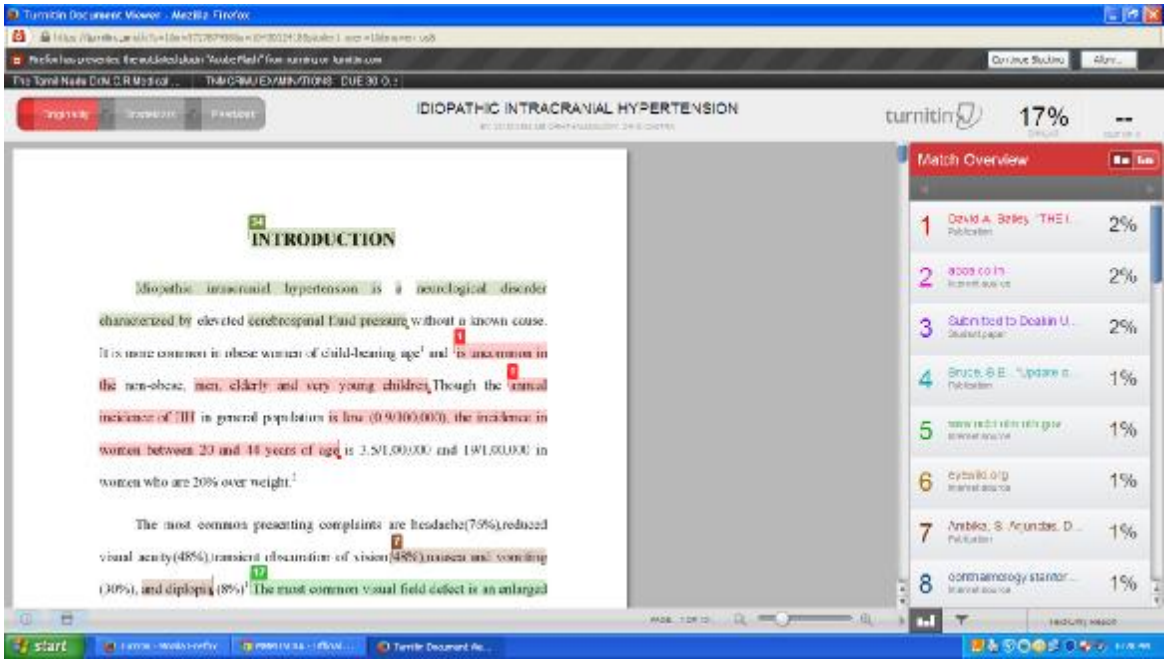
TSS - Transverse Sinus Stenosis

TREATMENT

DMX - Diamox

WR - Weight Reduction

IS - Iron Supplements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8 1%
INTRODUCTION
Idiopathic intracranial hypertension is a neurological disorder characterized by elevated cerebrospinal fluid pressure without a known cause. It is more common in obese women of child-bearing age[1] and is uncommon in the non-obese, men, elderly and very young children. Though the annual incidence of IIH in general population is low (0.9/100,000), the incidence in women between 20 and 44 years of age is 3.5/1,00,000 and 19/1,00,000 in women who are 20% over weight.[1]
The most common presenting complaints are headache(79%),reduced visual acuity(48%),transient obscuration of vision(44%),nausea and vomiting (30%), and diplopia (8%).[1] The most common visual field defect is an enlarged
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INTRODUCTION

MASTER CHART

S.NO	NAME	AGE	SEX	MR NO	DOV 1	DEF VISION	DURATION	HEADACHE	TOV	FIELD DEFECTS	VOMITING	DIPLOPIA	NECK PAIN	GIDDINESS	SYSTEMIC DS	TREATMENT H/O	PERSONAL H/O	WT IN KG	HT IN CM	BMI	BP in mmHg	BCVA RE	BCVA LE	PUPIL RE	PUPIL LE	EOM RE	EOM LE	FUNDUS RE	FUNDUS LE	IOP RE	IOP LE	CV RE	CV LE	CF RE	CF LE	Hb	RBS	LIPID PROFILE	TFT
1	Theen banu	24	F	393-0778	02.12.2014	2		1	1	2	2	2	1	2	HT, DYSLIP,	Anti-HT, OC	NIL	100	158	4	160/120	1	1	1	1	1	1	EP	EP	16	17	1	1	1	1	1	1	2	1
2	Prasanna	30	F	394-0142	15.12.2014	1	2	1	2	2	2	2	2	2	NIL	NIL	NIL	75	148	3	130/80	1	3	1	3	1	1	CP	CP	12	12	2	2	2	2	1	1	1	1
3	Singammal	25	F	151-4926	16.12.2014	2		1	1	2	2	2	1	2	NIL	NIL	NIL	56	140	2	110/70	1	1	1	1	1	1	EP	EP	17	16	1	1	1	1	2	1	1	1
4	Gnana vennila	22	F	394-2588	17.12.2014	2		1	2	2	1	1	1	2	NIL	NIL	NIL	52	146	1	110/70	1	1	1	1	2	1	EP	EP	15	12	1	1	1	1	1	1	1	1
5	Yuvanika	19	F	394-2776	18.12.2014	1	1	1	1	2	1	2	2	2	NIL	DOXY, VIT A	NIL	47	150	1	120/80	1	1	1	1	1	1	ESP	ESP	14	12	1	1	1	1	1	1	1	1
6	Vanitha	18	F	394-5956	23.12.2014	1	1	2	2	2	1	2	1	2	NIL	NIL	NIL	50	150	1	90/60	1	1	1	1	2	1	ESP	ESP	16	13	1	1	1	1	2	1	1	1
7	Sabeera barvin	29	F	392-4144	25.12.2014	2		1	1	2	2	2	2	2	PIH, PCOD	OCP	NIL	60	151	2	120/80	1	1	1	1	1	1	EP	EP	18	15	1	1	1	1	1	1	1	1
8	Pavithra	19	F	394-8265	26.12.2014	1	2	1	1	2	2	2	2	2	NIL	NIL	NIL	60	162	1	100/80	1	1	1	1	1	1	EP	EP	14	15	1	1	1	1	1	1	1	1
9	Joselin amala	21	F	395-0433	29.12.2014	2		1	2	2	2	2	2	2	NIL	NIL	NIL	55	153	1	110/70	1	1	1	1	1	1	EP	EP	12	12	1	1	1	1	2	1	1	1
10	Senthil kumar	32	M	360-8608	07.01.2015	2		1	2	2	1	2	2	2	NIL	NIL	NIL	65	158	2	110/70	1	1	1	1	1	1	EP	EP	11	14	1	1	1	1	1	1	1	1
11	Ruth renuga	34	F	395-8323	12.01.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	76	156	3	140/90	1	1	1	1	1	1	EP	EP	15	15	1	1	1	1	1	1		1
12	Chitra	35	M	395-8713	13.01.2015	2		1	2	2	1	2	1	1	NIL	NIL	NIL	51	148.5	1	110/80	1	1	1	1	1	1	EP	EP	16	14	1	1	1	1	1	1	1	1
13	Ravi	29	M	396-0210	16.01.2015	1	1	1	2	2	1	1	1	2	NIL	NIL	NIL	54	145	2	120/90	1	1	1	1	1	2	EP	EP	16	14	1	1	1	1	1	1	1	1
14	Podhu ponnu	31	F	149-3398	19.01.2015	1	2	1	2	2	1	2	2	2	NIL	NIL	NIL	62	150	2	90/50	1	1	3	1	1	1	ESP	ESP	12	12	2	2	1	1	2	1	1	2
15	Annapoorani	24	F	396-3453	22.01.2015	2		2	1	2	2	2	2	2	NIL	NIL	NIL	73	145	3	130/70	1	1	1	1	1	1	ESP	EP	10	13	1	1	1	1	1	1	1	1
16	Jamila banu	35	F	357-2883	24.01.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	69	152	3	120/80	1	1	1	1	1	1	EP	EP	17	16	1	1	1	1	2	1	2	1
17	Jeyalakshmi.A	49	F	396-4860	24.01.2015	2		1	1	2	2	2	2	2	NIL	NIL	NIL	70	155	2	100/70	1	1	1	1	1	1	ESP	ESP	12	13	2	1	2	1	1	1	1	1
18	Chinnanthi	36	F	396-5380	26.01.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	86	153	4	130/90	1	1	1	1	1	1	EP	EP	14	18	1	1	1	1	1	1	1	1
19	Uma maheswari	45	F	396-6047	26.01.2015	1	2	1	2	2	1	2	2	1	NIL	NIL	NIL	68	151	2	128/80	1	1	1	1	1	1	EP	EP	14	18	1	1	1	1	2	1	1	1
20	Emimal florance	37	F	396-5606	26.01.2015	2		1	1	2	2	2	2	2	NIL	NIL	NIL	54	152	1	100/60	1	1	1	1	1	1	ESP	ESP	11	14	1	1	1	1	2	1	1	1
21	Meher nisha	30	F	377-9916	27.01.2015	1	2	2	1	2	2	2	2	2	NIL	NIL	NIL	83	153	4	120/80	1	1	1	1	1	1	ESP	ESP	15	15	1	1	1	1	2	1	1	2
22	Jayalakshmi.M	43	F	396-9019	31.01.2015	2		1	1	2	2	2	2	2	DM,HT,THY	Anti-DM,Ar	NIL	66	154	2	150/90	1	1	1	1	1	1	ESP	ESP	15	14	1	1	1	1	2	1	2	1
23	Valarmathi	36	F	397-0511	03.02.2015	1	1	2	2	2	2	2	2	2	HT	Anti-HT	NIL	55	149	1	110/70	1	1	1	2	1	1	ESP	ESP	16	16	1	1	1	1	2	1	1	1
24	Saraswathi	34	F	397-1704	04.02.2015	2		1	1	2	2	2	2	2	NIL	NIL	NIL	70	150	3	130/80	1	1	1	1	1	1	EP	EP	12	12	1	1	1	1	2	1	1	1
25	Gayathri	29	F	397-2146	05.02.2015	2		1	2	2	1	2	2	2	NIL	NIL	NIL	71	155	2	110/80	1	1	1	1	1	1	ESP	ESP	15	14	1	1	2	2	1	1	1	1
26	Saranya	17	F	397-2113	05.02.2015	2		1	2	2	1	2	1	2	NIL	NIL	NIL	64	148.5	2	100/60	1	1	1	1	1	1	EP	EP	12	14	1	1	1	1	1	1	1	1
27	Bharathi priya	21	F	397-4634	09.02.2015	2		1	1	2	1	2	1	1	NIL	NIL	NIL	50	145.5	1	110/80	1	1	1	1	1	1	ESP	ESP	10	12	1	1	1	1	2	1	1	1
28	Bashurun	32	F	397-4728	10.02.2015	2		1	1	2	1	2	2	2	NIL	NIL	NIL	84	158.5	3	110/70	1	1	1	1	1	1	ESP	ESP	12	12	1	1	2	2	1	1	1	2
29	Vaishnavi	16	F	397-7805	14.02.2015	2	2	1	1	2	1	1	1	2	NIL	NIL	NIL	70	149	3	100/60	1	1	1	1	1	1	EP	EP	15	12	1	1	1	1	2	1	1	1
30	Bharathi	28	F	379-5064	14.02.2015	1		1	1	2	2	2	2	2	NIL	NIL	NIL	65	149	2	120/80	1	1	1	1	1	1	EP	EP	15	14	1	1	1	1	2	1	1	1
31	Uma	17	F	398-0827	18.02.2015	1		1	2	2	1	2	2	2	NIL	NIL	NIL	64	151	2	110/70	1	1	1	1	1	1	ESP	ESP	15	14	1	1	1	1	1	1	1	1
32	Murugalakshmi	26	F	398-4407	24.02.2015	2		1	1	2	2	1	2	2	NIL	NIL	NIL	62	143	3	120/80	1	1	1	1	1	2	ESP	ESP	15	13	1	1	1	1	1	1	1	1
33	Guruvammal	45	F	398-4800	25.02.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	61	150.5	2	140/80	1	1	1	1	1	1	CP	CP	18	19	1	1	1	1	2	1	1	1
34	Pandiarajan	41	M	398-5842	26.02.2015	2		1	1	2	2	2	2	2	HT	Anti-HT	NIL	90	170.5	3	140/100	1	1	1	1	1	1	EP	EP	15	15	1	1	1	1	2	1	1	1
35	Rajeshwari	24	F	398-9194	03.03.2015	1	1	1	1	2	2	2	2	2	NIL	NIL	NIL	68	154	2	120/60	1	1	2	2	1	1	ESP	ESP	14	16	1	1	1	1	2	1	1	1
36	Bala nagammal	41	F	399-0304	05.03.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	85	158	2	132/80	1	1	1	1	1	1	EP	EP	13	14	1	1	1	1	1	1	1	1
37	Sumangali	31	F	399-4548	12.03.2015	2		1	2	2	1	1	2	2	NIL	NIL	NIL	55	150	1	130/90	1	1	2	2	1	2	ESP	ESP	15	14	1	1	1	1	2	1	1	1
38	Mariyasugapriya	22	F	400-0215	20.03.2015	2		1	2	2	2	1	2	2	NIL	NIL	NIL	62	143	3	100/80	1	1	1	1	1	1	ESP	ESP	12	11	1	1	2	2	1	1	1	1
39	Radhalakshmi	32	F	399-6176	14.03.2015	2		1	1	2	2	2	2	1	NIL	NIL	NIL	67	158	2	130/80	1	1	1	1	1	2	ESP	ESP	16	18	1	1	1	1	1	1	1	1
40	Thavaselvi	36	F	399-6526	14.03.2015	2		1	1	2	1	2	2	2	THYROID	THYROXINE	NIL	71	156	2	110/70	1	1	1	1	1	2	ESP	ESP	18	20	1	1	2	2	2	1	1	2
41	Muthulakshmi	24	F	400-1764	23.03.2015	2		1	2	2	1	2	1	2	NIL	NIL	NIL	63	151	2	110/70	1	1	2	2	1	1	EP	EP	11	12	1	1	1	1	1	1	1	1

42	Subbulakshmi	26	F	400-3277	25.03.2015	1	1	1	2	2	1	2	1	2	NIL	NIL	NIL	54	149	1	120/80	1	1	1	1	1	1	ESP	ESP	18	19	2	2	2	2	2	1	1	1
43	Bhuvaneshwari	24	F	400-6407	31.03.2015	2		2	2	2	2	1	2	2	THYROID	ELTROXIN	NIL	62	148	2	110/70	1	1	1	1	1	2	ESP	ESP	14	15	1	1	1	1	2	1	1	1
44	Deepak venkat kumar	25	M	400-9347	04.04.2015	2		1	1	2	2	2	2	2	NIL	NIL	NIL	90	177	2	130/90	1	1	2	1	1	1	ESP	ESP	15	14	1	1	1	1	1	1	1	1
45	Pandeeswari	25	F	400-9403	04.04.2015	2		1	1	2	2	2	2	1	NIL	NIL	NIL	87	146	4	140/90	1	1	1	1	1	1	EP	EP	16	14	1	1	1	1	1	1	1	1
46	Ramya	27	F	401-0077	06.04.2015	2		1	2	2	1	2	2	2	NIL	NIL	NIL	85	158	2	110/80	1	1	1	1	1	1	EP	EP	15	16	1	1	1	1	1	1	1	1
47	Kaladevi	32	F	402-1727	23.04.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	54	148	1	120/80	1	1	2	2	1	1	ESP	ESP	18	18	1	1	1	1	1	1	1	1
48	Meena.D	42	F	401-1919	08.04.2015	1	2	2	2	2	2	2	2	2	NIL	NIL	NIL	100	148	4	140/80	1	1	1	1	1	1	EP	EP	10	18	1	1	1	1	2	1	1	1
49	Karuppayee	49	F	401-2808	09.04.215	2		1	2	2	1	2	1	2	HT	Anti-HT	NIL	90	152	4	114/84	1	1	1	1	1	1	ESP	ESP	14	15	1	1	1	1	1	1	2	1
50	Nasriya	27	F	401-5055	13.04.2015	1	1	2	2	2	2	2	2	2	HYPOTHYR(	THYRONOR	NIL	71	160	2	130/90	1	1	1	1	1	1	EP	EP	16	20	1	1	1	1	1	1	1	1
51	Ezhilarasi	28	F	401-5556	14.04.2015	1	1	1	2	2	1	1	1	2	NIL	NIL	NIL	64	148	2	90/70	1	1	2	2	1	1	ESP	ESP	14	14	1	1	1	1	1	1	1	1
52	Nagajothi	23	F	401-5503	14.04.2015	2		1	1	2	2	2	1	2	NIL	NIL	NIL	50	155	1	130/70	1	1	2	2	1	1	ESP	ESP	10	13	1	1	1	1	1	1	1	1
53	Dhanalakshmi	18	F	402-1932	23.04.2015	2		1	2	2	1	1	2	2	NIL	NIL	NIL	70	148	3	140/90	1	1	2	2	1	2	ESP	ESP	11	12	1	1	1	1	2	1	1	2
54	Meena.M	39	F	402-5664	29.04.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	78	162.5	2	120/70	1	1	1	1	1	1	EP	EP	16	17	1	1	1	1	2	1	1	1
55	Murugeswari	36	F	403-4873	12.05.2015	2		1	1	2	2	2	2	2	NIL	NIL	NIL	68	153	2	130/90	1	1	1	1	1	1	ESP	ESP	12	12	1	1	1	1	2	1	1	1
56	Durga	31	F	403-7246	15.05.2015	2		1	2	1	2	2	2	2	NIL	NIL	NIL	87	150	4	150/100	1	1	1	1	1	1	ESP	ESP	12	13	1	1	2	2	2	1	1	1
57	Sangeetha	25	F	403-8714	16.05.2015	1	1	1	1	2	1	2	2	2	NIL	NIL	NIL	64	148	2	130/70	1	1	1	1	1	1	ESP	ESP	13	12	1	1	1	1	1	1	1	1
58	Bharathi	31	F	404-0473	19.05.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	65	149	2	110/70	1	1	1	1	1	1	ESP	ESP	16	15	1	1	1	1	2	1	1	1
59	Pandi selvi	49	F	404-6761	26.05.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	50	151	1	150/80	1	1	1	1	1	1	EP	ESP	12	14	1	1	1	2	1	1	1	1
60	Anandhajothi	28	F	404-9132	29.05.2015	2		1	2	2	2	2	2	2	NIL	NIL	NIL	60	152	2	110/70	1	1	1	1	1	1	ESP	ESP	12	12	1	1	1	1	1	1	1	1
61	Renugadevi	24	F	404-9756	30.05.2015	1	1	1	2	2	2	2	2	2	NIL	NIL	NIL	65	148.5	2	110/70	1	1	1	1	1	1	ESP	ESP	11	14	1	1	2	2	1	1	1	2

CT BRAIN	MRI	MRV	HFA 30-2	TREATMENT	DOV 2	WT IN KG	V/A RE	V/A LE	PUPIL RE	PUPIL LE	FUNDUS RE	FUNDUS LE	CV RE	CV LE	CF RE	CF LE	TREATMENT	DOV 3	WT IN KG	V/A RE	V/A LE	PUPIL RE	PUPIL LE	FUNDUS RE	FUNDUS LE	CV RE	CV LE	CF RE	CF LE	TREATMENT	
	1 and 2	normal	2	WR,DMX																											
	1 and 2	normal	2	WR,DMX	22.01.2015	74	1	1	2	3	CP and OA	CP and OA	2	2	2	2	DMX														
1 and 2			2	WR,DMX	05.01.2015	56	1	1	1	1	RP	RP	1	1	1	1	DMX	23.02.2015	56	1	1	1	1	N	N	1	1	1	1	DMX	
	1 and 2	normal	1	DMX	24.01.2015	52	1	1	1	1	RP	RP	1	1	1	1	DMX														
	1 and 2	normal	1	DMX	13.02.2015	47	1	1	1	1	RP	RP	1	1	1	1	DMX														
	1 and 2	normal	1	DMX,IS	08.01.2015	55	1	1	1	1	RP	RP	1	1	1	1	DMX,WR														
1	1	HLTS,HLSS	2	DMX,WR	10.10.2015	60	1	1	1	1	CP	CP	1	1	1	1	DMX,WR														
	1	normal	1	DMX																											
	1	normal	1	DMX	22.01.2015	55	1	1	1	1	RP	RP	1	1	1	1	DMX	13.02.2015	54	1	1	1	1	N	N						
	1	normal	2	DMX,WR	07.08.2015	65	1	1	1	1	N	N	1	1	1	1	WR														
	1 and 2	normal	1	DMX,WR	19.02.2015	75	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	27.03.2015	75	1	1	1	1	N	N						
	1 and 2	HLTS,HLSS	1	DMX	20.02.2015	50	1	1	1	1	N	N	1	1	1	1															
	1	normal	1	DMX,WR	18.02.2015	52	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	13.03.2015	52	1	1	1	1	N	N						
1 and 2			1	DMX,WR	02.02.2015	62	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	05.03.2015	52	1	1	1	1	N	N	1	1		1	DMX,IS	
	1 and 2	normal	1	DMX,WR	20.02.2015	73	1	1	1	1	RP	RP	1	1	1	1	DMX,WR														
1	1 and 2	normal	2	DMX,WR,IS	26.02.2015	68	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	19.03.2015	67	1	1	1	1	N	N						
1			2	DMX	02.03.2015	70	1	1	1	1	RP	CP	1	1	1	1	DMX	08.03.2015	68	1	1	1	1	N	N	1	1	1	1	WR	
	1 and 2	normal	2	DMX,WR	08.08.2015			1	1	1	N	N	1	1	1	1	WR														
	1 and 2	normal	1	DMX, IS	12.03.2015	68	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	17.04.2015	67	1	1	1	1	N	N	1	1	1	1	WR	
	1 and 2	normal	2	DMX	02.03.2015	51	1	1	1	1	ESP	ESP	1	1	1	1	TOPIRAL	04.04.2015	54	1	1	1	1	RP	RP	1	1	1	1	DMX	
	1 and 2	HLTS,HLSS	2	DMX,WR,IS	10.02.2015	83	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	07.03.2015	82	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	
	1	HLTS,HLSS,RTS,RSS	2	DMX,WR,IS	07.04.2015	64	1	1	1	1	N	N	1	1	1	1	WR,IS														
1	1 and 2	HLTS,HLSS,RTS,RSS	1	DMX,IS	27.03.2015	51	1	1	1	1	RP	RP	1	1	1	1	DMX,IS	10.06.2015	51	1	1	1	1	N	N	1	1	1	1	IS	
	1 and 2	B/L TSS	1	DMX,WR,IS	26.03.2015	70	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	15.05.2015	69	1	1	1	1	N	N	1	1	1	1	WR	
1 and 2			2	DMX,WR																											
	1 and 2	normal	1	DMX	26.03.2015	64	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	11.04.2015	64	1	1	1	1	N	N	1	1	1	1	WR	
	1	normal	1	DMX,IS	02.03.2015	50	1	1	1	1	ESP	ESP	1	1	1	1	TOPIRAL	08.06.2015	52	1	1	1	1	RP	RP	1	1	1	1	TOPIRAL	
	1 and 2	HRTS,HRSS,LTS,	2	ONSD	17.03.2015	78	1	1	1	1	CP	CP	1	1	1	1	DMX,WR.IS	01.05.2015	78	1	1	1	1	CP	CP	1	1	1	1	DMX,WR,IS	
	1	normal	1	DMX,WR,IS	25.02.2015	70	1	1	1	1	RP	RP	1	1	1	1	DMX,WR.IS														
	1 and 2	normal	1	DMX,WR,IS																											
	1 and 2	normal	1	DMX	19.03.2015	64	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	14.05.2015	62	1	1	1	1	N	N	1	1	1	1	WR	IS
1			2	DMX	13.03.2015	62	1	1	2	2	ESP	ESP	1	1	1	1	DMX	18.04.2015	62	1	1	1	1	RP	RP	1	1	1	1	DMX	
	1	normal	1	DMX	01.07.2015	60	1	1	1	1	RP	RP	1	1	1	1	DMX	19.08.2015	60	1	1	1	1	N	N	1	1	1	1	WR	
	1 and 2	normal	2	DMX,WR,IS	18.03.2015	88	1	1	1	1	RP	RP	1	1	1	1	DMX.WR.IS	30.04.2015	88	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	
1 and 2			1	DMX,WR	18.03.2015	68	1	1	2	2	ESP	ESP	1	1	1	1	DMX,WR														
1			2	DMX,WR	21.03.2015	85	1	1	1	1	EP	EP	1	1	1	1	DMX,WR	14.07.2015	83	1	1	1	1	N	N	1	1	1	1	WR,IS	
	1 and 2	LTS,LSS	2	DMX,IS	16.04.2015	55	1	1	1	1	RP	RP	1	1	1	1	DMX,IS	04.06.2015	55	1	1	1	1	N	N	1	1	1	1	IS	
	1	HLTS,HLSS	2	DMX,WR	18.04.2015	58	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	18.04.2015	58	1	1	1	1	N	N						
	1	HLTS,HLSS	1	DMX,WR	08.04.2015	66	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	13.06.2015	64	1	1	1	1	N	N	1	1	1	1	WR	
	1 and 2	HLTS,HLSS	2	DMX,WR,IS	21.03.2015	71	1	1	1	1	ESP	ESP	1	1	2	2	DMX,WR,IS	16.04.2015	70	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	
	1 and 2	normal	1	DMX,WR	18.04.2015	63	1	1	1	1	EP	EP	1	1	1	1	DMX,WR	15.04.2015	63	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	

1	1 and 2	HLTS,HLSS	2	MANNITOL	14.04.2015	54	1	1	2	2	RP	RP	1	1	1	1	DMX	25.05.2015	54	1	1	1	1	N	N	1	1	1	1	
1			2	DMX,WR,IS	17.04.2015	64	1	1	1	1	ESP	ESP	1	1	1	1	DMX,WR,IS	15.05.2015	61	1	1	1	1	RP	RP	1	1	1	1	DMX,WR.IS	
	1 and 2	HLTS,HLSS	2	DMX,WR	25.04.2015	95	1	1	1	1	ESP	ESP	1	1	2	2	DMX,WR	30.05.2015	95	1	1	1	1	RP	RP	1	1	2	2	DMX.WR	
	1 and 2	HLTS,HLSS	1	DMX,WR	24.04.2015	86	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	16.05.2015	86	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	
	1 and 2	HLTS,HLSS	1	DMX,WR	15.04.2015	87	1	1	1	1	N	N																		
	1 and 2	normal	1	DMX	25.05.2015	54	1	1	1	1	RP	RP	1	1	1	1	DMX	12.06.2015	53	1	1	1	1	N	N	1	1	1	1		
	1 and 2	normal	2	DMX,WR,IS	15.05.2015	98	1	1	1	1	RP	RP	1	1	1	1	TOPIRAL,WR	22.06.2015	98	1	1	1	1	RP	RP	1	1	1	1	DMX,WR.IS	
	1 and 2	HLTS,HLSS	1	DMX,WR	16.05.2015	90	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	20.06.2015	90	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	
	1	RTS	1	DMX,WR	14.05.2015	70	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	26.06.2015	70	1	1	1	1	N	N						
	1	normal	1	DMX,WR	27.04.2015	64	1	1	1	1	ESP	ESP	1	1	1	1	MANNITOL	25.06.2015	64	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	
	1 and 2	HLTS,HLSS	2	DMX	18.05.2015	50	1	1	1	1	ESP	ESP	1	1	1	1	DMX	16.05.2015	50	1	1	1	1	RP	RP	1	1	1	1	DMX	
	1	HLTS,HLSS	2	DMX,WR,IS	07.05.2015	70	1	1	1	1	ESP	ESP	1	1	1	1	DMX,WR,IS	02.06.2015	70	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	
	1	HLTS,HLSS	2	DMX,WR,IS	20.05.2015	76	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	23.07.2015	75	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	
	1 and 2	LTS	2	DMX,WR,IS	11.07.2015	68	1	1	1	1	RP	RP	1	1	1	1	DMX,WR,IS	18.08.2015	67	1	1	1	1	N	N	1	1	1	1		
	1 and 2	normal	2	DMX.WR,IS	07.07.2015	87	1	1	1	1	ESP	ESP	1	1	1	1	DMX,WR,IS	18.08.2015	85	1	1	1	1	ESP	ESP	1	1	1	1	DMX,WR,IS	
	1	HLTS,HLSS	2	DMX,WR	03.06.2015	60	1	1	1	1	ESP	ESP	1	1	1	1	DMX,WR	13.07.2015	60	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	
	1	HLTS,HLSS	1	DMX,WR,IS	25.06.2015	65	1	1	1	1	RP	RP	1	1	1	1	DMX.WR.IS														
	1	normal	2	LP	27.04.2015	50	1	1	1	1	RP	RP	1	1	1	1	DMX	28.05.2015	50	1	1	1	1	N	N	1	1	1	1	DMX	
	1 and 2	normal	1	DMX,WR	23.06.2015	60	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	29.07.2015	60	1	1	1	1	N	N	1	1	1	1	WR	
	1	normal	2	DMX,WR	25.06.2015	65	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	30.07.2015	63	1	1	1	1	RP	RP	1	1	1	1	DMX,WR	